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Contents

Page

1 Introduction 1

2 HPA Annual Air Pollution Research Lecture 3

A few things you didn't know about air pollution and health 3

3 Keynote Addresses 5

3.1 PM–opsonin interactions: Implications for airborne PM and nanoparticles 5

3.2 Air pollution and health: New approaches to older issues and further challenges 7

3.3 Discussion 8

4 Atmospheric Chemistry, Exposure Measurement and Modelling 9

4.1 Model development and validation of personal exposure to VOC concentrations (MATCH Project) 9

4.2 Changes in second-hand smoke concentrations in bars following smoke-free legislation in Scotland and England 13

4.3 An investigation of the effect of external flow and pollution concentration fields on the infiltration of pollution into naturally ventilated buildings 17

4.4 An improved model for indoor air chemistry 19

4.5 An overview of indoor contamination with persistent organic pollutants 22

4.6 Discussion 25

5 Particles and Health 27

5.1 Alteration of fibrin clot properties by ultrafine particulate matter 27

5.2 Relation between respiratory symptoms and lung function in rural and urban Nepalese adults 28

5.3 Science and communication: Does ultrafine particulate matter from waste incineration affect infant mortality? 31

5.4 Particulate air pollution and risk of stillbirth: The UK PAMPER study, 1961–92 35

5.5 Discussion 38

6 Epidemiology, Public Health and Policy Development 39

6.1 Links between urban ambient particulate matter and health: Time series analysis of particle metrics 39

6.2 Quantification for health impact assessments: Linking deaths, lives and values 40

6.3 Modelling solvent dispersion, chemistry and health impacts to inform policy development on VOC control 42

6.4 An evaluation of the impact of the congestion charging scheme on pollution concentrations in London 43

6.5 Waste derived fuels in cement kilns, public health implications and response: Assessing the evidence 44

6.6 Discussion 46

7 Parallel Discussion Sessions **47**

7.1 Particle toxicology 47

7.2 Distributed lags in epidemiology: Should we be using distributed lags for quantification? 50

8 Posters **55**

8.1 Vapour:particle phase distribution of hexabromocyclododecane (HBCD): Are PUF disk passive samplers suitable for monitoring HBCD in indoor air? 55

8.2 Tetrabromobisphenol-A (TBBP-A) in air and dust from Birmingham, UK: Implications for human exposure 59

8.3 Tools for smoke deposition measurement in the human lung 63

8.4 Correlation between microenvironment and personal exposure levels of nitrogen dioxide: Research and policy implications 65

8.5 Intercomparison of particulate matter monitoring devices 69

8.6 Breach of the NO_2 standard due to roadwork-caused traffic congestion and associated abuse of the protocol on NO_2 diffusion tubes 73

8.7 Constructing and validating modelled concentration surfaces for black smoke and sulphur dioxide across GB, 1955–2001 78

8.8 Nanoparticles in urban air: A very small problem 81

8.9 Oxidative capacity of ambient PM_{10} collected at an industrial waste transfer facility 85

8.10 Bioreactivity of airborne PM_{10} particulate matter from a municipal solid waste landfill 89

8.11 Reactive oxygen species drives coal fly ash bioreactivity 94

8.12 The effect of particulate air pollution on birth weight: Results from the UK PAMPER study 98

8.13 Brominated flame retardants in dust: Implications for human exposure 102

8.14 Methodological approaches to examining joint effects of noise and air pollution on health outcomes in populations living near major airports: Study design 105

8.15 Arrhythmogenic and ischemic effects of particulate air pollution on patients with cardiac disease: Preliminary analysis of ECG parameters 108

8.16 Size distribution of fungal and bacterial aerosol in domestic environment 112

9 Closing Remarks **115**

Annex I Workshop Participants **117**

Annex II Workshop Programme **119**

1 Introduction

This, the eleventh in a series of annual review meetings on outdoor and indoor air pollution research in the UK, was hosted by the Institute of Environment and Health (IEH) on behalf of the Department of Health (DH) and Health Protection Agency (HPA). It is the second such meeting hosted by the Institute since its re-establishment at Cranfield University in November 2005. As with previous meetings, the principal intention was to facilitate the exchange of information between the various UK-based research groups and between researchers and those involved in policy and regulatory development, and to stimulate discussion of pertinent research issues in the fields of atmospheric chemistry, exposure measurement, modelling, risk assessment and the characterisation of hazard potential (including the study of mechanisms of toxicity and epidemiology). The proceedings of a number of previous meetings have been published and are available to download from the IEH website (IEH, 2000, 2002, 2004a/b, 2005, 2007).

This year's meeting was particularly memorable for the inclusion of the inaugural lecture of the newly established HPA Annual Air Pollution Research Lecture series. This lecture, entitled A few things you didn't know about air pollution and health, was given by Professor Bert Brunekreef of the Institute for Risk Assessment Sciences (IRIS), Utrecht University, The Netherlands (see Section 2). Two further invited keynote presentations – on particle surface interactions and new approaches to epidemiological studies on air pollution and health – were given by Dr Michaela Kendall and Professor Klea Katsouyanni, respectively (Section 3). The remainder of the meeting was structured around a series of topic-orientated sessions (Sections 4–6) and a twin parallel discussion session (Section 7). In a few instances, for example because the presentations included preliminary or as yet unpublished information, it has not been possible to include abstracts in this report. In these cases only a brief outline of the scope of the presentation is given.

A number of posters were also presented at the workshop; abstracts are provided in Section 8. Finally, a summary of the closing remarks by Dr Peter Rombout of the National Institute for Public Health and Environment, The Netherlands, and the associated discussions, are presented in Section 9.

The meeting opened with a welcome from Professor Robert Maynard CBE of the HPA, who noted the continued importance of research into the contribution of air pollutants to adverse health effects in human populations. Professor Maynard praised the DH for their efforts to maintain an active and successful UK air pollution research programme, and noted the recent developments within the HPA in establishing a programme of research targeted at furthering our understanding of the emerging hazards associated with the rapid expansion in the applications of nanotechnology and nanomaterials and the consequent increased risk of exposure to such novel materials.

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2 HPA Annual Air Pollution Research Lecture

Professor Maynard introduced the 2008 HPA Annual Air Pollution Research Lecture, emphasising the exceptional contributions made to this field by the first individual to be awarded this distinction, Professor Brunekreef.

A few things you didn't know about air pollution and health

B Brunekreef

IRIS, University of Utrecht, The Netherlands

In a wide ranging presentation, Professor Brunekreef discussed the history of our developing understanding of the interactions between human health and atmospheric pollution. The long standing nature of many of today's challenges was noted; for instance, road dust is still an issue in many developed and developing countries but was first publicly reported as a health concern in 1896, when the focus was on high levels of horse-drawn traffic. Attention was drawn to the little-recognised global nature of the threat now posed by some sources of particles, such as Saharan dust storms which are capable of causing pollution episodes across Europe. Also, it was noted that such particle sources can result in exposures to both biological and non-biological agents that are capable of exerting adverse health effects. Professor Brunekreef noted that levels of air pollution have fallen dramatically since the 1960s. However, the initial optimism that exposure was being reduced below effect thresholds has faded as the results of various time-series studies had overturned the initial simplistic notions that thresholds of effect operate at the population level. There is thus a continuing need to research the impacts of air pollution on health, and many challenges remain.

During subsequent discussion it was asked whether the coarse fraction of the ambient aerosol contributes to the effects of roadside exposure to air pollution on health. Professor Brunekreef noted that the literature indicated that particles arising from brake linings and other road-related dusts may contribute to the coarse fraction and may include toxic constituents.

3 Keynote Addresses

Invited keynote presentations were given on *PM–Opsonin interactions: Implications for airborne PM and nanoparticles* by Dr Kendall and *Air pollution and health: New approaches to older issues and further challenges* by Professor Katsouyanni.

3.1 PM–opsonin interactions: Implications for airborne PM and nanoparticles

Michaela Kendall

University of Birmingham, UK, & Uludag University, Turkey

Evidence from two methodological approaches – epidemiology and toxicology – has shown detrimental health changes in response to atmospheric exposures to fine and nano-sized particulate matter (PM; Pope and Dockery, 1999). Implicated PM characteristics include size, composition and surface area (Oberdörster *et al*., 1994; Peters *et al*., 1997). The exact mechanism(s) by which inhaled nano-sized particles may affect the lungs and heart, however, remains unknown. We do know that PM with small aerodynamic diameters – including nanoparticles and PM of specific length-to-diameter ratios or low density – access the lower lung in greater quantities than PM with larger aerodynamic diameters. In the alveolar spaces these particles initially impact on the surfactant lung-lining liquid layer. This layer enables oxygen exchange, lung expansion, and also provides primary host defence against depositing foreign material such as solid and biological particles. Particles may then translocate to the circulatory system and other organs (Oberdörster *et al*., 2005).

Initial and continued PM interactions with the body occur at PM surfaces. Inhaled PM surface area and these particles' inherent 'stickiness' may therefore be important factors in determining PM effects. Opsonisation, where constituents of serum coat foreign bodies and promote phagocytosis, could be involved. We are currently testing the hypothesis that opsonisation of deposited PM by collectins in the lung alters the downstream behaviour of invading particles, thereby impeding normal lung and serum performance. It has yet to be shown whether opsonisation is as important in the labelling and collection of solid particles as for biological particles, but several studies have demonstrated parallels (Kendall *et al*., 2001, 2002, 2004a, 2004b and 2007, amongst others). The most significant health consequences may therefore be expected where large surface areas of relatively surface-active materials are deposited, which can be observed in some epidemiological and toxicological studies. Since the coating of deposited PM by host opsonins may modulate their biological activity, as they do for infectious particles, physical studies of such interactions provide new information on how inhaled PM may induce pulmonary and cardiovascular toxicity. When combined with clinical studies, such knowledge may identify biological mechanisms and will also help in predicting hazard and risk associated with synthetic nanoparticle exposures, some of which may have similar health effects to atmospheric PM.

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3.2 Air pollution and health: New approaches to older issues and further challenges

Klea Katsouyanni & Evangelia Samoli

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The study of the health effects of outdoor air pollution is one of the most active research areas of environmental epidemiology. To date, we can say that there is an overall consensus about the magnitude of the short-term effects, that is, an increase in the daily number of deaths of about 0.5 to 0.6% per 10 $\mu g/m^3$ in PM_{10} concentration, and a somewhat greater increase for cardiac/cardiovascular and respiratory mortality, for a one to two-day exposure, whilst estimates for $PM_{2.5}$ effects are mainly based on US studies and are more variable. The effect appears to increase when exposure over more days or months is taken into account.

The effects of long-term exposure to air pollution, using particle measurements or gaseous indices of traffic pollution, indicate a substantial increase in total mortality (by 6 to 17% per 10 $\mu g/m^3$ $PM_{2.5}$ or NO_2) and cardiopulmonary mortality (by 9 to 28%, respectively). Recent results from the American Cancer Society (ACS) reanalysis for Los Angeles (Jerrett *et al.,* 2005) and from a cohort in women (Miller *et al.,* 2007) indicate that estimated effects are larger when within-city $PM_{2.5}$ concentrations are contrasted rather than between-city pollution levels.

Many questions remain concerning the characteristics of PM associated with their toxicity. It appears that coarse particles have a different range of effects compared to fine particles, but this requires further study. There is accumulating indirect and more direct evidence that particles originating from traffic are associated with larger effects than those from other sources.

With regard to methodology, geographic information systems (GIS), used to estimate spatial variability in air pollution and long-term exposure, are now also applied to produce 24-hour estimates and may even be extended to finer time scales.

There has been evidence in recent years that sensitive subgroups for air pollution effects are the elderly, people with co-morbidities, and, for some outcomes, children. Some of these impacts have been refined and understood better. Also, two papers on atherosclerosis have shed light on early effects of air pollution.

Since the last WHO revision of the air quality guidelines there has been renewed interest in gaseous pollutants, with new Air Pollution and Health: a European Approach (APHEA) and other publications on NO_2, CO and ozone which show that we should not abandon the attempt to understand the complex relationship between gases and particles and their independent health effects.

Finally, last year's extensive forest fires in Greece and California have attracted considerable publicity, and in this context results from an analysis on the effects of forest fires from an earlier time period have proved useful in assessing health risks to exposed populations.

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3.3 Discussion

Dr Kendall was asked about the reversibility of particle aggregation in the bloodstream and the use of inhibitors to prevent aggregation. It was noted that whilst aggregation does occur, it has only been examined at 24-hour time intervals, time points which are too far apart to determine inhibitory or reversible effects. Furthermore, it was noted that the pH in the lung is within the range pH 6.6–6.8 but the experiments had been done at pH 7.

In the discussions that followed Professor Katsouyanni's lecture it was noted that the effects identified appeared to relate to long-term exposure, which varied from five to twenty-five year time scales. The nature of the heart rate modifying effects was, however, still an issue for ongoing analyses, with both O_3 and PM levels possibly showing correlations but only in the summer period; there was no apparent confounding between the two factors (ozone and PM_{10}). The question was asked if, from the data presented, the 85+ years of age subgroup represented a 'spared' subpopulation. In response, it was noted that the degree of effect seen differed between the various pollutants and that this might suggest that differences in the underlying toxic mechanisms of the different pollutants are being detected.

4 Atmospheric Chemistry, Exposure Measurement and Modelling

Under the chairmanship of Dr Stuart Harrad of the Univeristy of Birmingham, five papers were presented relating to the fields of exposure measurement and modelling, and environmental chemistry.

4.1 Model development and validation of personal exposure to VOC concentrations (MATCH Project)

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****Presenter***

Background and objectives

The term 'air toxics' is used in relation to a range of substances associated mainly but by no means exclusively with emissions from road vehicles. Whilst a number of adverse health outcomes may be associated with a number of air pollutants, the most important, especially in the public mind, is likely to be cancer. Currently, however, evidence for carcinogenicity derives very largely from epidemiological studies of occupationally exposed individuals, or even from laboratory studies with animal models. In the case of pollutants such as benzene and the polycyclic aromatic hydrocarbons, where the evidence for carcinogenicity deriving from occupational exposures is very strong, the exposures greatly exceed the typical environmental concentrations to which the general public are exposed, and therefore evaluation of carcinogenic risk other than through extrapolation is very difficult.

This proposal is concerned primarily with the issue of differentiating personal exposures to air toxic substances according to documented exposure to emissions from specific sources, such as road traffic and environmental tobacco smoke (ETS). The aim is to provide a validated approach to personal exposure estimation such that exposure can be estimated from a lifestyle questionnaire, allowing differentiation of individuals into a range of personal exposure groupings which could be used in a subsequent epidemiological study of either a case–control or an ecological (spatial analysis) design.

Since 'air toxic' substances do not arise uniformly from a single source, their concentrations may not correlate between different exposure environments and therefore it is desirable to measure microenvironment concentrations of a wide range of such substances to establish their inter-relationship, while also determining the relationships between microenvironment concentrations and personal exposure by direct measurements and modelling.

Overall aim and specific goals of research

The overall aim of the study was to quantify the magnitude and range of individual personal exposures to a range of air toxics and to develop models for exposure prediction based upon time/activity diaries.

The specific goals of the research were as follows.

- To use personal monitoring of non-smoking volunteer subjects with a range of residential locations and exposure to non-traffic sources to assess daily exposures
- To determine microenvironment concentrations of a range of 'air toxic' substances, taking account of spatial and temporal variations and hotspots
- To optimise a model of personal exposures based upon microenvironment concentration data and time/activity diaries and to cross-compare modelled exposures with exposures independently estimated from personal monitor data
- To produce a scheme for categorising exposure (by compound) according to location of residence and other lifestyle and exposure factors (e.g. ETS) for use in design of case–control and ecological studies of cancer incidence

Study description

Personal exposure measurement

The targeted air toxics were volatile organic compounds and polycyclic aromatic hydrocarbons, as follows.

- Volatile organic compounds (VOC): benzene, ethylbenzene, n-hexane, naphthalene, styrene, toluene, o-xylene, m-xylene, p-xylene, 1,3,5-trimethylbenzene, 1,2,4-trimethylbenzene, p-isopropyltoluene, pyridine, 3-ethenylpyridine and 1,3-butadiene
- Polycyclic aromatic hydrocarbons (PAH): acenaphthylene, acenaphthene, fluorene, phenanthrene, anthracene, fluoranthene, pyrene, benzo(a)anthracene, chrysene, benzo(b)fluoranthene, benzo(j+k)fluoranthene, benzo(a)pyrene, indeno(1,2,3-cd)pyrene, benzo(ghi)perylene, dibenzo(ah)anthracene, coronene

Using actively pumped personal samplers, volunteers collected 24-hour integrated personal samples during 5 days for VOC compounds and for 1 day for PAH compounds. The sampling method (Kim *et al*., 2001) to measure VOC (excluding 1,3-butadiene) involved drawing air through an adsorbent tube packed with Tenax GR followed by Carbotrap collecting 57.6 L of VOC sample. To measure 1,3-butadiene, 43.2 L of air was pumped through an adsorbent tube packed with Carbopack B followed by Carbosieve SIII. Particle phase polycyclic aromatic hydrocarbons were collected onto a pre-treated quartz fibre filter sampling 4.3 m^3 of air (Harrison *et al*., 2007).

Microenvironment measurement

During the period when personal exposure was being measured there was a simultaneous programme of measurement of workplace and home environments, as well as a seasonal programme of measurement of other microenvironments that the subjects visited during their daily activities. These other microenvironment measurements included street microenvironments (e.g. trafficked roadside locations, background streets, parks), transport microenvironments (e.g. cars, trains, bus, coach stations), indoor environments (e.g. restaurants, libraries) and other home environments (e.g. garden, garages, spare bedrooms).

Using specifically designed pumped microenvironment samplers, samples were collected for shorter time periods (e.g. 12 hours for homes, 8 hours for offices and 2 hours for other microenvironments) for the same compounds. The same volumes of air were collected for microenvironment samples for

VOC and 1,3-butadiene, 57.6 L and 43.2 L respectively. For PAH microenvironment samples collected from homes and offices the same amount of air (4.3 m^3) was collected, but for other microenvironments samples a lower volume of PAH sample was collected (1.44 m^3) due to technical restrictions in sampling flow rates.

Analysis

Analysis of VOC (excluding 1,3-butadiene) was by means of a thermal desorber (Tekmar 6000/6016) interfaced with a gas chromatograph and mass selective detector. Chromatographic separation was achieved using a CP-wax 52 CB capillary column (50 m, 0.25 mm id, 0.20 µm film thickness). Full details of conditions for the desorption and gas chromatography appear in our earlier paper (Kim *et al.*, 2001).

Analysis of 1,3-butadiene used a thermal desorber interfaced with a gas chromatograph and mass selective detector. Chromatographic separation was achieved using a CP-PoraBOND Q capillary column (50 m, 0.32 mm id, 5 µm film thickness).

Particle-phase PAH collected onto pre-treated quartz fibre filter were extracted with solvent, purified and concentrated prior to analysis with a gas chromatograph and mass selective detector. Chromatographic separation was achieved using a HP5-ms capillary column (30 m, 0.25 mm id, 0.25 µm film thickness).

Personal exposure modelling

Volunteers were asked to list in a diary their activities each day, to describe the places they visit and the journeys they take. With this information it has been possible to reconstruct exposures to 'air toxics' based upon the location information and time–activity records.

The personal exposure data has been split in two different and independent datasets. The first set contains 75% of the data and was used for training the model. The other 25% of the data was saved as contrast dataset in order to check and validate the model developed with the training dataset and to verify its reliability in predicting personal exposures.

The model proposed predicts the personal exposure, integrating the time fraction spent in each microenvironment times the concentration of each microenvironment visited and accounting also for external factors that might affect as add-on variables, as shown in Equation 1.

$$P_{ij} = \alpha \cdot \sum \frac{t_{ijk} \cdot C_k}{T_{ij}} + \sum \beta_m A_m + \sum \gamma_n F_n \qquad (1)$$

- P_{ij} is the personal exposure for a subject i on a day j
- t_{ijk} is the time spent in microenvironment k by subject i on a day j
- C_k is the concentration representative of each microenvironment k visited by subject i
- T_{ij} is the total time spent in all different microenvironments for subject i on a day j
- α is the coefficient associated with the first term of the equation
- A_m are different explanatory variables describing activities performed on a day j by a subject i or characteristics associated to a volunteer i
- β_m is the coefficient associated to the explanatory variable A_m
- F_n represents the time spent in doing different activities
- γ_n is the coefficient associated to the factor F_n

The explanatory variables, A_m, have a value of 1 if the activity is performed or characteristic is present, or 0 on the contrary. The variables representing the time spent in doing different variables, F_n, were measured in minutes. A total of 112 add-on variables, A_m and F_n, extracted from the collected information were included into the model. The model was developed using the Stepwise option from the Linear Regression menu in the SPSS 15.0 for Windows.

Results

The variability of VOC and PAH personal exposure concentrations mainly reflects the range of activities the subjects engaged in during the 5-day period of sampling, as well as the variability in ambient and indoor levels, which is due to varying environmental conditions.

It appears that personal exposure levels are in reasonable agreement with 'home' concentrations. Furthermore, personal exposure concentration remained consistently higher during days of the week associated with increased vehicle use and ETS exposure.

Regarding microenvironment concentrations, indoor microenvironments generally showed higher concentrations than outdoor microenvironments. The highest concentrations for both VOC and PAH were recorded in those microenvironments with ETS events. Outdoor microenvironment concentration reflected the effect of traffic.

The model developed reflects the VOC concentrations, explaining high levels of variance, for example 80% for ethylbenzene, xylenes, styrene and trimethylbenzenes and around 50% for compounds such as benzene, toluene and 1,3-butadiene.

The developed models were tested in the validation dataset and there is good agreement between the measured and the predicted values for most of the compounds, except for naphthalene and 1,3-butadiene. This conclusion is supported by the correlation coefficient (R) for plots of direct versus indirect exposure estimates tested in a separate and independent validation dataset, which span from 0.4 for benzene to 0.9 for styrene.

Conclusions and discussion

The results from the sampling campaign have shown personal exposure patterns that are consistent with the associated microenvironmental concentrations and subject lifestyles.

Models have been developed and validated for predicting personal exposure to VOCs.

Acknowledgments

This study was conducted as part of the MATCH Project (Measurement and Modelling of Exposure to Air Toxic Concentrations for Health Effect Studies). The funding for this project was provided by the Health Effects Institute. We would like to thank all the subjects who participated in this study. The authors are especially grateful to Dr Ben Armstrong for his extensive advice on statistical issues.

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4.2 Changes in second-hand smoke concentrations in bars following smoke-free legislation in Scotland and England

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****Presenter***

Background and objectives

Exposure to second-hand smoke (SHS) increases the risk of developing lung cancer (Hackshaw *et al*., 1997) and cardiovascular disease (Law *et al*., 1997) among non-smokers. Public health policy in many countries has moved to control non-smokers' exposure to SHS, with recent legislation introduced across the UK and many parts of Europe to prohibit smoking in enclosed public places. Scotland introduced the Smoking, Health and Social Care (Scotland) bill to ban smoking in public places from 26 March 2006 with England introducing almost identical measures via the Health Act (2006) on 1 July 2007.

Workers in the hospitality sector have among the highest exposures to SHS of all occupational groups (Howard, 2004). Studies have shown that non-smoking bar workers have salivary cotinine levels of four times the level of non-smokers who live with partners who smoke, and almost ten times the levels of non-smokers living in non-smoking households (Jarvis, 2001). A review of data on exposure to SHS across a wide range of entertainment establishments (Siegel & Skeer, 2003) indicated that airborne nicotine concentrations were up to 18.5 times higher than in offices or domestic residences. These occupational exposures to such high levels of indoor air pollution are likely to have resulted in chronic disease in many individuals, with one analysis by Jamrozik (2005) suggesting that about 54 hospitality workers die every year in the UK as a result of their exposure to SHS. Many more will suffer from acute and chronic respiratory symptoms as a result of their workplace SHS exposure.

NHS Health Scotland undertook an extensive evaluation of the Scottish smoke-free legislation and the Bar Workers' Health and Environmental Tobacco Smoke Exposure (BHETSE) study was one element of this work (Haw *et al*., 2006). The Department of Health also commissioned a comprehensive evaluation programme to investigate the effects of the introduction of the smoke-free legislation in England in July 2007. Building on experience from the BHETSE study, the Smoke-free Bars 07 project was designed, led by the University of Aberdeen in collaboration with the Institute of Occupational Medicine and Liverpool John Moore's University.

Both studies had the following objectives.

- Examining changes to bar workers' respiratory and general health
- Analysing changes in attitudes to SHS and smoke-free legislation
- Measurement of changes in bar air quality before and after implementation of smoke-free legislation

This paper only reports the findings relating to the air quality objective.

Study description

Both the BHETSE and Smoke-free Bars 07 studies were longitudinal in design with a three phase follow-up of a random selection of bar workers from pre-selected geographical areas across Scotland and England. The first phase was carried out in the months leading up to the implementation of the

smoke-free legislation, the second phase between 2 and 3 months post-implementation, and the third phase at approximately 1-year after the collection of Phase 1 data. The Scottish study ran from January 2006 until February 2007. The English study began in May 2007 and completes Phase 3 in May 2008. As a result only data looking at changes between Phase 1 and 2 is presented here.

In Scotland, bars were recruited from three city areas (Aberdeen, Edinburgh and Glasgow) and two rural settings (Aberdeenshire and Borders). In England the areas selected included three urban areas (central London, Newcastle-upon-Tyne and Liverpool) and two rural areas (Northumbria and Cumbria). These areas were selected primarily for logistical reasons but also with consideration for covering a wide range of socio-economic and geographical areas.

Bar workers' exposure to SHS was assessed by three main methods. Two methods (discreet/area and overt/personal) using measurement of airborne levels of particulate matter less than 2.5 μm in size ($PM_{2.5}$) and the third by measurement of bar workers' saliva concentrations of cotinine, a metabolite of nicotine. Details of the Scottish overt/personal exposure data are presented in Semple *et al*. (2007a). This paper presents the discreet/area sampling results and salivary cotinine data from Phase 1 and 2 of both studies.

Discreet measurement of $PM_{2.5}$

Our methodology for the discreet sampling is described in full in Semple *et al.* (2007b). In brief it involved the use of a battery operated aerosol monitor (TSI SidePak AM510 Personal Aerosol Monitor) fitted with an impactor in order to sample the concentration of $PM_{2.5}$. The monitor was placed in a small bag with a short length of Tygon tubing attached to the inlet and left protruding to the outside. The monitor was zero calibrated each day prior to use and the airflow rate set at 1.7 l/min using a Drycal DC Lite flow meter. The monitor was switched on to log at 1-minute intervals at least 5 minutes prior to entry to a pub.

On entry to each establishment the researcher purchased a beverage before proceeding to a seat or area as central as possible and away from any doors, windows or obvious potential sources of $PM_{2.5}$ such as open solid-fuel fires or kitchen areas. The researcher aimed to place the bag containing the monitor at seat or table level, to ensure that sampling was as close to the breathing zone as possible, and also tried to ensure that no smoking was occurring within 1 metre of the sample collection point. Air sampling was carried out for a minimum of 30 minutes. Data from each pub visit was downloaded to PC using the TSI TrackPro v3.41 software. A calibration factor of 0.295 was applied to all raw measurements to correct for the properties of SHS particles. A time-weighted $PM_{2.5}$ average concentration was calculated for each visit.

Return follow-up visits at Phase 2 were carried out on the same day of the week and within ± 30 minutes of the timing of the baseline visit.

Salivary cotinine

Collection of a sample of saliva from each study participant and analysis of the concentration of cotinine was carried out using methods described in full in Semple *et al.* (2007a). In brief, at all three survey visits a non-stimulated saliva sample was collected from participants using a salivette (Sarstedt Ltd, Leicester). Samples were sent to ABS Laboratories, London, and analysed for cotinine using a previously published method using a rapid gas–liquid chromatographic technique (Feyerabend and Russell, 1990). Results less than the limit of detection (LOD, 0.1 ng/ml) were assigned a value of half the LOD. For the purposes of analysis we compared the self-report of smoking behaviour (never, ex-, regular and occasional smoker) with the salivary cotinine level. A cut-off of 20 ng/ml salivary cotinine has been indicated as the level above which active smoking is likely to have taken place and we used this concentration in creating sub-groups of confirmed smokers/non-smokers.

Results

The BHETSE study visited a total of 41 bars at Phase 1 and Phase 2; some bars were visited twice to gather data at quiet and busy times. The English Smoke-free Bars 07 study visited 62 bars at Phase 1 and 52 at Phase 2. BHETSE recruited 371 bar workers at baseline and followed up 266 of these at Phase 2. The on-going English study involved 180 at Phase 1, with 118 being seen again at Phase 2.

The discreet sampling undertaken in both studies showed marked reductions in $PM_{2.5}$ concentrations, as illustrated in Table 4.2.1. Of the 53 pairs of visits carried out in Scotland, all bars showed reductions in $PM_{2.5}$ concentrations after the ban, while 51 of the 52 venues in England were lower at Phase 2 compared to Phase 1.

Table 4.2.1 Changes in $PM_{2.5}$ levels in bars between Phase 1 and 2

	England $PM_{2.5}$ levels (μg/m³) (n = 52 visits)		**Scotland $PM_{2.5}$ levels (μg/m³) (n = 53 visits)**	
	Phase 1	Phase 2	Phase 1	Phase 2
$PM_{2.5}$ GM	81	7	167	16
$PM_{2.5}$ AM	140	11	246	20

GM, geometric mean; AM, arithmetic mean

Table 4.2.1 shows reductions in $PM_{2.5}$ – a marker for SHS concentrations – between Phase 1 and Phase 2 in both the English study and the earlier BHETSE study in Scotland. The scale of the change in both countries is very similar, with a fall of about 91% in the geometric mean figure in England compared to 90% in Scotland by the same methodology.

Table 4.2.2 presents comparison of salivary cotinine among smoking and non-smoking bar workers in both studies at Phase 1 and Phase 2.

Table 4.2.2 Bar workers' salivary cotinine levels* between Phase 1 and Phase 2

	England cotinine (ng/ml)		**Scotland cotinine (ng/ml)**	
	Phase 1	Phase 2	Phase 1	Phase 2
Non-smokers	1.47	0.36	2.94	0.70
Smokers	223	178	207	209

* Geometric mean data

Both studies show reductions in salivary cotinine levels among non-smoking bar workers of about 76%. Non-smoking bar workers in England had baseline levels about half those of their Scottish counterparts – broadly confirming the finding from the discreet $PM_{2.5}$ measurements that English bars had airborne SHS levels of about one-half of those found in Scottish bars at Phase 1.

By Phase 2, non-smoking bar workers in England had salivary cotinine levels similar to those found in the general adult population (0.35 ng/ml), reflecting their marked reduction in exposure at work but their continued exposure at home, outside and in social settings in other people's homes.

The change in the salivary cotinine level among bar workers who smoke in England is perhaps suggestive of changes in smoking habits. Smoking bar workers in Scotland did show reductions in their smoking behaviour of about 1.7 cigarettes per day by Phase 3.

Discussion and conclusions

Average $PM_{2.5}$ concentrations in bars were high before the introduction of smoke-free laws. In Scotland the average $PM_{2.5}$ level was nearly four times the US EPA 'unhealthy' level (65 μg/m^3) for

$PM_{2.5}$ in outdoor air. In English bars the average $PM_{2.5}$ level was over twice this level. Some bars had 30-minute levels that were over 15 times the 'unhealthy' index concentration.

Baseline levels of $PM_{2.5}$ were about 40% higher in Scotland than England – and cotinine measurements in non-smoking bar workers support this finding. The reasons for this are unclear. A proportion of this difference may be due to the timing of the Phase 1 baseline surveys in each country. Phase 1 was carried out in the winter months in Scotland, when people would have been smoking inside the bars. Phase 1 in England was carried out in May and June, when the weather was more pleasant and bar customers may have been sitting in open-air areas where available. Other reasons for the difference may include the lower population smoking prevalence in England compared to Scotland, differing cultural attitudes to smoking, better ventilation in English pubs and a shift in the 'acceptability' of smoking in the presence of non-smokers between 2006 and 2007.

Before the introduction of smoke-free legislation, non-smoking bar workers in England had salivary cotinine levels of approximately five times the UK population average, while bar workers in Scotland had levels nearly ten times the population average.

Smoke-free legislation in both Scotland and England has led to marked reductions in $PM_{2.5}$ concentrations in bars. The magnitude of the improvement within two months of implementation in both countries seems to have been broadly similar with reductions in $PM_{2.5}$ levels of about 90%. Post-ban $PM_{2.5}$ levels in bars are now in the range of those found in outside ambient air in most cities.

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4.3 An investigation of the effect of external flow and pollution concentration fields on the infiltration of pollution into naturally ventilated buildings

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**Presenter*

Background and objectives

The need for filtration of pollution in urban buildings close to major sources of pollution (roadways) carries large energy penalties that might not repay later in terms of indoor air quality and occupant comfort. Previous studies have reported that naturally ventilated buildings offer little or no protection against infiltration of external pollution. However, infiltration of air from outside is directional, depending on the air flow routes the building envelope affords and the distribution of driving pressures around the building. The performance of prediction tools appropriate to building ventilation design has not been demonstrated for this application in complex urban topography.

Study description

Indoor and outdoor concentrations of carbon monoxide (CO) were measured in three naturally ventilated (NV) spaces and two mechanically ventilated (MV) spaces facing onto the same heavily trafficked central London road. The concentration distribution around the building was sampled, in addition to local meteorology above-roof and in the street. Key findings were as follows.

Results

Indoor/outdoor (I/O) concentration ratios were highly variable with wind direction in the NV spaces, and could be as low as 0.3 when the street-side façade is leeward. Variations in I/O ratios with wind direction and speed were regular and in good agreement with basic ventilation relationships that underpin models commonly employed in practice to predict air infiltration for ventilation design, indicating that the variation is predictable.

However, external concentrations in the street and at the façade vary considerably from point to point, as does the relationship between concentrations in the roadway (source) and at the façade. The position of the external sensor in this context is a significant source of variation or uncertainty in the determination of I/O ratios.

Conclusions

Simple adjustments to infiltrations models could be made to account for directionality of infiltration to (a) improve accuracy of exposure analysis for urban naturally ventilated buildings and (b) to assist design for a more protective envelope.

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4.4 An improved model for indoor air chemistry

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Background and objectives

It is well established that some atmospheric pollutants are damaging to health, with their outdoor concentrations subject to regulation. However, despite the fact that urban dwellers in developed countries have been estimated to spend ~90% of their time indoors (home, commuting, office), indoor air quality has received relatively little attention. The combined effect of indoor sources of pollutants and infiltration of pollutants generated outdoors leads to the concentrations of some pollutants being higher indoors than out, with concentrations high enough to have an adverse impact on health. The latter observation has led to a UK government advisory group concluding that exposure to pollutants generated outdoors is likely to largely occur indoors (COMEAP, 2004).

There are many sources of indoor pollution. Pollutants can be generated through indoor activities such as cooking (particles, carbon monoxide, nitrogen oxides, NO_x), cleaning (volatile organic compounds, VOCs, such as monoterpenes) and smoking (carbon monoxide, particles, NO_x and VOCs), whilst others are emitted from building, furnishing and consumer products such as carpets, adhesives, paints, houseplants and toiletries (Crump *et al*., 1997; Yu & Crump, 1998; Wolkoff *et al*., 2000; Nazaroff & Weschler, 2004). In addition, some indoor pollutants are formed outdoors but make their way inside through doors, windows or ventilation systems, or infiltrate the fabric of the building.

Although there is increasing research being carried out on indoor air quality, there are still many unknowns (Carslaw, 2003). Most research until recently has focused on species that are emitted indoors and relatively little attention has been paid to the significance of the reactions between them. In this paper the chemical reactions taking place in a typical residential house in the UK are investigated in detail using a photochemical box model. Unlike previous indoor air chemistry modelling studies (Nazaroff & Cass, 1986; Weschler & Shields, 1996; Drakou *et al*., 1998; Sarwar *et al*., 2002; Sørensen & Weschler, 2002) the model contains an explicit chemical mechanism, with no lumping of chemical reactions and no surrogate species. Therefore, it is possible to investigate in detail the chemical reactions responsible for driving the indoors chemistry.

Study description

A detailed chemical box model has been constructed based on the Master Chemical Mechanism (MCMv3.1), a comprehensive mechanism that treats explicitly the atmospheric degradation of 135 VOCs (Jenkin *et al.*, 2003; Saunders *et al.*, 2003). The degradation of each VOC is initiated by reaction with OH and, where appropriate, direct photolysis and reactions with ozone and the nitrate radical. The species generated following initiation processes can each undergo a number of further reactions. Each product is degraded in turn, resulting eventually in the final degradation products CO_2 and H_2O. The MCM also contains a comprehensive inorganic scheme, including the chemistry of O_3, NO_x and carbon monoxide. The MCM was modified for indoor air, resulting in the addition of 2000 new reactions describing deposition, emissions, exchange, surface reactions and additional gas-phase chemistry (Carslaw, 2007). The model has also been updated recently to include gas to particle conversions for α- and β-pinene and limonene.

The room is assumed to have an area to volume (A/V) ratio of 3.0 m^{-1} for the base case scenario, typical for many furnished homes (Wainmann *et al.*, 2001) and an air exchange rate of 2 ach^{-1} (Carslaw, 2007). The temperature of the room was constant at 293 K with a relative humidity of 50%. The model was run for three days to allow steady-state to be achieved and all reported results are for the third day of the model runs. The base case model results are for 21June and 50°N.

Results

The results show a predicted indoor OH radical concentration up to 4.0×10^5 molecule cm^{-3} (Carslaw, 2007). Although the daytime value is lower than that outdoors in summer (typically 2×10^6 molecule cm^{-3}), it is comparable with daytime values in the winter (typically 5×10^5 molecule cm^{-3}; Emmerson *et al.*, 2005) and with night time values of OH observed outdoors (1×10^5 to 1×10^6 molecule cm^{-3}) (Faloona *et al.*, 2001; Platt *et al.*, 2002). These results are in reasonable agreement with past studies (Weschler & Shields, 1996, 1997; Sarwar *et al.*, 2002) and support the notion that OH radicals can reach appreciable quantities indoors. The results also show that there is a wide range of particle products formed indoors, and that the formation of these particles is sensitive to a number of model input parameters such as lighting levels, air exchange rate and indoor activities.

Conclusions

This study has highlighted that it is possible to have significant concentrations of OH indoors, comparable to those observed outdoors at night time and during the day in winter. Radical chemistry indoors is more important than might have been imagined given the much lower levels of light compared with outdoors. Indeed, initiation, termination and propagation reactions appear to occur at similar rates to suburban environments outdoors in the summer, but with a greater emphasis on the oxidation of monoterpenes and alkenes by ozone. Measurements of OH indoors are vital to back-up model predictions.

The particle concentrations indoors are also of concern, particularly given their extremely small size, which makes them likely to end up in the human respiratory system if inhaled.

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4.5 An overview of indoor contamination with persistent organic pollutants

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**Presenter*

Background and objectives

Until recently, human exposure to persistent organic pollutants (POP) had been widely considered to occur almost exclusively via the diet. While this appears true for dioxins, it may not be the case for those POPs with indoor use patterns. This paper summarises our research on the causes, levels and human exposure implications of indoor contamination by polychlorinated biphenyls (PCB) and a number of brominated flame retardants (BFR), in particular polybrominated diphenyl ethers (PBDE), and hexabromocyclododecanes (HBCD). Human health concerns have been raised with respect to each of these chemical classes, creating a need for exposure assessment. While manufacture and new use of PCBs ceased in the UK in the late 1970s, there is an unknown but likely substantial proportion of the ~40 000 t UK usage remaining in use in applications such as window sealants. BFR usage is also substantial, with the (recently banned) Penta-BDE commercial formulations deployed widely in polyurethane foam used in furniture. In addition, substantial manufacture continues of the Deca-BDE product for flame-proofing of textiles and high-impact polystyrene (HIPS) housing for electronic goods such as TVs, as well as of HBCD for use in building insulation, and HIPS. In addition to direct exposure via inhalation and ingestion of indoor air and dust, there are concerns that such indoor contamination has implications for future dietary exposure, either via emissions from indoor environments during use of treated materials, or as a result of their disposal at end-of-life (Harrad and Diamond, 2006). Given the vast quantities of materials treated with BFRs at per cent levels that will require disposal, this constitutes an issue with substantial implications for sustainable chemicals management.

Study description

Over the last few years, our research group has conducted a number of studies recording concentrations of PCBs, PBDEs, and HBCDs in indoor air and dust from domestic, office, and vehicular microenvironments, as well as outdoor air and soil. The relationships between indoor and outdoor contamination have also been explored through comparison of chiral signatures of PCBs. Our sampling and chemical analysis methods are described in detail elsewhere (Harrad *et al*., 2006; Jamshidi *et al*., 2007; Abdallah *et al*., 2008).

Results

Contamination of indoor air and dust

Table 4.5.1 summarises concentrations of PCBs, PBDEs and HBCDs measured in UK indoor air between 2003 and 2007. For all target contaminants, substantial indoor:outdoor increments exist. Concentrations of PBDEs and HBCDs in UK indoor dust over the period 2005 to 2007 are summarised in Table 4.5.2. Based on these data, inhalation contributes 4.2–63% of overall adult exposure to PCBs, but is less significant for PBDEs and HBCDs. For both PBDEs and HBCDs dust ingestion is more important, with the highly skewed distribution of concentrations meaning that for some individuals (especially toddlers who are assumed to ingest more dust), it constitutes the principal pathway of exposure, far exceeding that received via the diet. For HBCDs, some toddlers appear to be exposed at levels consistent with those experienced by occupationally exposed adults.

Table 4.5.1 Concentrations (pg m^{-3}) of ΣPCB, ΣPBDE, and ΣHBCD in domestic indoor air

Statistical parameter/ Compound	PCB[a]	PBDE[b]	HBCD[c]
Average	2800	52	250
σ_n	2600	61	240
Median	1800	24	180
Min	490	4	67
Max	9800	250	1300

[a] sum of tri-hepta chlorinated PCBs; [b] sum of tri-hexa brominated PBDEs; [c] sum of α-, β-, and γ–HBCD

Table 4.5.2 Concentrations (ng g^{-1}) of ΣPBDE and ΣHBCD in UK domestic indoor dust

Statistical parameter/ Compound	PBDE[a]	HBCD[b]
Average	260 000	6000
σ_n	580 000	20 000
Median	8500	730
Min	12	140
Max	2 200 000	110 000

[a] sum of tri-deca brominated PBDEs; [b] sum of α-, β-, and γ-HBCD

Contamination of outdoor air and soil

During 2003–2004, monthly time-weighted average atmospheric concentrations of PCBs and PBDEs were measured over a calendar year at ten sites located on a 79 km rural:urban transect in the prevailing wind direction across the West Midlands. Concentrations in matched soil samples were also recorded. Figure 4.5.1 illustrates the concentration trends in air for PBDEs, with a clear 'urban increment' evident. Similar results were observed for soil and for PCBs. Concentrations of HBCDs in indoor air (average 250 pg m^{-3}) substantially exceed those recorded in five samples of outdoor air on the University of Birmingham campus (average 37 pg m^{-3}).

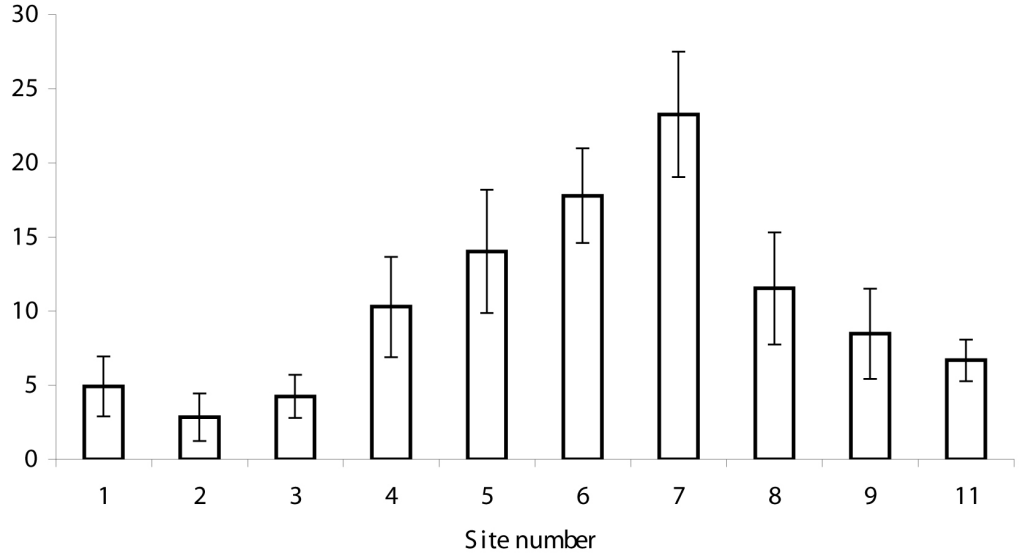
Chiral signatures of PCBs in indoor air, outdoor air and soil

Chiral signatures of PCB 95 in both indoor and outdoor air are universally racemic. This contrasts with the non-racemic signatures detected in most soil samples.

Conclusions

- Concentrations of PCBs in indoor air in the West Midlands exceed substantially those in outdoor air and have not declined significantly from those recorded in indoor air by our group in 1997 (Currado & Harrad, 1998).
- The similarity between chiral signatures of PCB 95 in indoor and outdoor air (but not soil) suggests strongly that emissions from the built environment – even 30 years after the ban on manufacture and new use – are driving contemporary concentrations in outdoor air. This is consistent with the observed urban increment in concentrations of PCBs.
- The importance of indoor contamination as a source of outdoor contamination of recent and current use BFRs is illustrated by the urban increment observed for PBDEs.

Figure 4.5.1 Variation in concentrations of PBDEs in outdoor air along a rural:urban transect (error bars are $\pm 1\sigma_n$). Site numbers are from 1 (rural southwest) through city centre (Site 7) to 11 (rural northeast)



Discussion

Contamination of indoor environments with PCBs, PBDEs and HBCDs constitutes a substantial source of human exposure, and measures to control such exposures and to evaluate their health impacts appear prudent. The absence of a temporal decline in PCB contamination of indoor air since 1997 coincides with a similar stagnation in UK human dietary exposure to non-dioxin-like PCBs. This suggests that tackling continuing indoor contamination is essential if human exposure is to be reduced further. Of wider significance, the fact that PCB exposure remains substantial three decades after the cessation of their manufacture and new use suggests that similar policies aimed at limiting exposure to BFRs and related chemicals are unlikely to be sufficient, and that measures to manage the end-of-life of materials incorporating such chemicals are required.

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4.6 Discussion

With regard to the study on the effects of a smoking ban on exposure in Scotland presented by Professor Ayres, it was asked whether there had been any issues in gaining ethical approval for the study, given the essentially 'covert' methods used to obtain atmospheric exposure measures. It was noted that this had not been considered an issue by the local ethical committee.

The presentation of a model to predict indoor air chemistry interactions, given by Dr Carslaw, provoked considerable interest. The relative contribution of indoor generated particles to overall exposures was questioned, given that there is known to be a potential for considerable ingress of outdoor particles. In response, Dr Carslaw noted that internal generation may account for up to a quarter to a third of total exposures, so is potentially of significance. The sensitivity of the model to uncertainties in reaction kinetics was queried. It was noted that the Master Chemical Mechanism model includes between 12 and 13 thousand reactions, of which only approximately 10% have actually been demonstrated and characterised; the rest are predicted using structure and property-based computational techniques. The existence of a degree of uncertainty was, therefore, implicit in the model design. The influence of light levels and the role of deposition indoors are, however, likely to be of greater relevance than any model limitations with regard to rate coefficients; the dataset on the propagation of light within domestic and office situations, and indeed our knowledge of light light-driven reactions, were noted to be limited in extent. A participant noted that in future it would be worth considering whether it would be possible to incorporate model approaches developed by architects to predict light propagation within buildings. It was also suggested that this study highlighted the need to combine the existing 'traditional' exposure models, which address only primary particle exposures, with indoor chemistry models of this type that would allow prediction of secondary generated particle levels.

With regard to Dr Harrad's presentation, it was noted that there was evidence from the USA suggesting a link between the presence of PCB in dust and in human milk, highlighting the potential for uptake from environmental sources.

5 Particles and Health

Under the chairmanship of Professor Terry Tetley, Imperial College, London, four papers were presented on the subject of airborne particles and their consequences for human health.

5.1 Alteration of fibrin clot properties by ultrafine particulate matter

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This presentation discussed studies undertaken *in vitro* to investigate the characteristics of blood clot formation in response to the presence of atmospheric particles of various sizes, including ultrafine particles. A particular focus of the work related to the possible formation of abnormal clot structures in the presence of such particulate matter. However, at the request of the author no detailed abstract is available for inclusion in this report.

5.2 Relation between respiratory symptoms and lung function in rural and urban Nepalese adults

*OP Kurmi, M Steiner, S Semple & JG Ayres**

Department of Environmental and Occupational Medicine, University of Aberdeen

Presenter

Background and objectives

Indoor smoke exposure is a recognised major cause of ill health in less economically developed countries (LEDC; Pandey *et al*., 1989; Smith *et al*., 2000; Smith, 2003). Recent estimates indicate that around half of the world's population, more than three billion people, use unprocessed biomass fuels such as wood, dung, crop residues and coal to meet their basic household energy demand (Bruce *et al*., 2002; Rehfuess, 2006). It is estimated that the world's total energy consumption from biomass has decreased from 50% in 1900 to a current approximate value of 14% (Bruce *et al*., 2002; Parikka, 2004), but in more than 30 countries, primarily those that are less economically developed, wood still provides more than 70% of household energy requirements. Biomass is the major cooking fuel in rural Nepal, whereas the urban Nepalese population uses fuels such as liquefied petroleum gas (LPG) and kerosene. The number of households that use solid fuel as their main energy source in Nepal is close to 85% (Government of Nepal, 2006). Exposure to biomass smoke is associated with chronic obstructive pulmonary disease, chronic bronchitis, asthma and respiratory infections (Campbell *et al*., 1989) as well as non-respiratory effects such as cataract, although in many studies confounding factors have been poorly or not addressed and in few have direct measures of exposure been undertaken.

Respiratory symptoms and lung function measurements were carried out in a study of rural and urban populations in Nepal to assess the relationship between measured exposure to emissions from different types of fuel and respiratory and cardiovascular health

Study description

We report a cross-sectional study of two rural areas in the Kathmandu valley, and an urban area, Kathmandu Metropolitan City, Nepal. An investigator-delivered questionnaire gathered details of smoking history, socio-economic status, ventilation and structure of kitchen, fuel history and respiratory and cardiovascular health symptoms in rural and urban adults aged 16 or over in 490 houses. In addition, spirometry was measured using EasyOne spirometers. 24-hour indoor particulate matter ($PM_{2.5}$) and 12-hour outdoor concentrations (using DustTrak and SidePak light scattering devices), respirable particulate matter and 24-hour temperature, relative humidity and carbon monoxide levels were measured in the kitchen.

Households were selected using cluster random sampling in both urban and rural areas. 245 households in the rural area and an equal number of households from the urban area were sampled. Prior to sampling each household was visited personally to obtain written, informed consent for the measurements to be taken. PM sampling was done in households that used firewood or LPG as their main fuel in the rural and urban areas, respectively, and where at least two adults (greater or equal to 16 years of age) were living.

Results

The findings presented below relate to the results of preliminary analyses. Detailed multivariate analyses have yet to be undertaken.

Exposures

The 24-hour mean concentrations of indoor $PM_{2.5}$ in rural and urban households were 690 $\mu g/m^3$ and 140 $\mu g/m^3$, although peaks in excess of 30 000 $\mu g/m^3$ were regularly seen during cooking in the rural areas. The 24-hour mean concentrations of outdoor $PM_{2.5}$ for the rural and urban households were 198 $\mu g/m^3$ and 178 $\mu g/m^3$ respectively, the rural sources being from disturbed dust and smoke from indoor fuel burning. Prevalence of current smoking was 11.2% in the urban area compared with 30.3% in the rural area, with around 10% in both areas reporting having smoked in the past.

Demographics

48.8% of the 846 rural dwellers and 47.3% of the 802 urban dwellers were male. Mean age for the rural population was 36 years (range 16 to 98) and for the urban population 35 years (range 16 to 87).

Lung function

Overall, 180/1228 (14.7%) of those with acceptable spirometry of all subjects fulfilled the GOLD criteria for COPD (any severity), but the prevalence was higher in the rural (18.9%) compared to the urban (10.9%) populations. 58/311 (18.6%) of rural women and 34/339 (10%) of urban women had COPD of any severity The figures for rural men and urban men were 52/274 (19%) and 36/304 (11.8%) respectively.

Symptoms

Respiratory symptoms generally increased with age. Wheeze 'ever' was reported in 22.6% of all rural dwellers but only in 6.9% of all urban dwellers (OR 3.96, $p < 0.001$) while wheeze in the last 12 months was less common, although still with a rural predominance (12.7% vs 6.4%; OR 2.1, $p < 0.01$). Wheezing was more common in smokers than non-smokers, but more so in rural dwellers (rate ratios for current vs non-smokers being 3.73 rural and 2.14 urban).

The prevalence of morning cough was non-significantly higher in urban compared to rural dwellers (6.4% vs 5.2% respectively) while coughing phlegm on waking was four-fold higher in urban than in rural dwellers (15.6% vs 4.0% respectively, OR 0.23, $p < 0.001$). Again, smoking interacted but in a different way, rate ratios for morning phlegm (current vs non-smokers) being 2.56 and 3.23 for urban and rural dwellers respectively.

Breathlessness was more prevalent in the urban population, possibly partly related to their higher BMI (rural mean 20, range 11 to 31; urban mean 23, range 14 to 40) and lower level of fitness.

Symptoms in relation to COPD

24/82 (29.3%) of rural women who reported wheeze had COPD compared to 8/27 (29.6%) of urban women. For rural and urban males the respective figures were 21/50 (42%) and 5/17 (29.4%). However, COPD was also prevalent in those who did not report wheeze: 34/229, 14.9% for rural women; 26/312, 8.3% for urban women; 31/224, 13.8% for rural men; and 31/287, 10.8% for urban men). 21% of rural women and 13% of rural men reporting shortness of breath with wheeze had COPD compared to 4% of both urban women and men.

Discussion

Indoor $PM_{2.5}$ concentrations were higher in the rural households compared to the urban households, but reported smoking prevalence was low in the urban area. The high exposure to biomass smoke in the rural areas was associated with a greater prevalence of COPD, particularly in women who spend a considerable time in the kitchen doing the cooking and are thus exposed to biomass more than are urban women who use mixed types of cleaner fuels for cooking. Wheezing illness was much more common in the rural than in the urban population but, curiously, productive cough was much more common in the urban dwellers, which could be due to under-reporting of cigarette smoking (although

this would have to be considerable to reverse the pattern), be related to exposure to urban outdoor air pollution, or related to some other factor such as cooking fume or occupation. Many subjects reported respiratory symptoms with normal spirometry. We are now looking at measures of small airway flow in these populations, which may detect earlier changes in response to the relevant exposure, whether it be indoor biomass smoke, cooking fume itself or, in the urban population, outdoor air pollution.

These findings support work from elsewhere on the importance of biomass smoke exposure to respiratory health, but is one of the first to deal adequately with confounders. Current exposure to biomass smoke does not seem to predict proportionate loss of lung function, suggesting either that past exposures were different or that mass is an inappropriate marker of the key factor in smoke which drives the respiratory effects.

Interventions aimed at reducing indoor exposures by use of more efficient stoves and ducting smoke outdoors have been shown to be beneficial in terms of lung function in both China and Guatemala, but the mechanisms of these effects remain to be defined. As lung function differences are noticeable by the age of 16, attention needs to be paid to the effect on lung development.

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5.3 Science and communication: Does ultrafine particulate matter from waste incineration affect infant mortality?

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Background

Waste incineration facilities have been the subject of considerable public concern, opposition, fear, and even hatred for many years. Over the past two decades the key aspects of public concern have included:

- emissions of dioxins and furans
- emissions of metals
- increased cancer risk
- increased risk of birth defects
- adverse effect on recycling
- emissions of ultrafine particulate matter.

The UK is currently undertaking a major investment in waste treatment infrastructure. This includes the development of a wide range of facilities for sorting and treating waste, including combustion facilities.

This paper considers the current objections to waste incineration. Based on experience of addressing health concerns in relation to waste incineration, conclusions have been drawn in relation to the communication of science in a complex area.

Objections to waste incineration

At a public meeting in February 2008, 33 individual objections to waste incineration were raised. These included criticisms of previous research (e.g. Elliott *et al.*, 1996; Elliott *et al.,* 2000), comments relating to a range of health effects such as infant mortality and cancer, comments relating to pollutants including ultrafine particles (also referred to as $PM_{2.5}$), and recommendations for an alternative technology.

In the past two or three years, objectors to waste-to-energy facilities have increasingly placed an emphasis on ultrafine particulate matter. For example, 12 of the 33 points referred to above make explicit reference to ultrafine particulate matter, and concerns relating to ultrafine particulates are implicit in many of the other points.

These criticisms are extremely worrying, particularly for a lay audience. The local MP described these comments as 'frightening,' and a local councillor and decision-maker said that he was not previously aware of how bad incinerators are. The result is that local residents, decision-makers and other stakeholders are left with the impression that there must be a basis to some of the claimed problems, even if only because of the number of claims being made.

Those making these criticisms state that supporting evidence is available. For example, a sheet of over 300 references was provided at the public meeting in February 2008. However, a clear and detailed statement of the exact supporting evidence referred to is not readily available, either as a public presentation, a scientific publication, or via an online resource.

Those making these criticisms have the freedom to make wide-ranging assertions. Those representing or advising the waste industry do not have such freedom. So how can the waste industry ensure that these concerns are properly addressed, and provide a more balanced public debate than is currently taking place?

One approach is to look in more detail at each point being raised. The criticisms are made in very unambiguous terms; environmental issues associated with waste management facilities are typically complex and cannot be readily expressed in such simple terms. This indicates that the criticisms as presented are simplifications. By making these simplifications, the detailed science behind each criticism may have been lost or distorted.

Case study: Infant mortality

Point 2 above and recent media reports refer to a study of infant mortality. This research is claimed to demonstrate that the rate of infant mortality is significantly higher downwind of waste incinerators than the rate upwind of waste incinerators. Studies of infant mortality rates in a number of areas have been produced – see Figure 5.3.1 for example, which shows the infant mortality rate in the wards around the Coventry waste-to-energy facility. In Figure 5.3.1, the wards are grouped into an 'upwind' group (green) with a relatively low infant mortality rate, and a 'downwind' group (red) with a relatively high infant mortality rate.

The same data are presented in more detail in Figure 5.3.2, which shows the infant mortality rates for each individual ward.

Figure 5.3.1 Infant mortality rates in the vicinity of the Coventry waste incinerator (upwind and downwind areas)

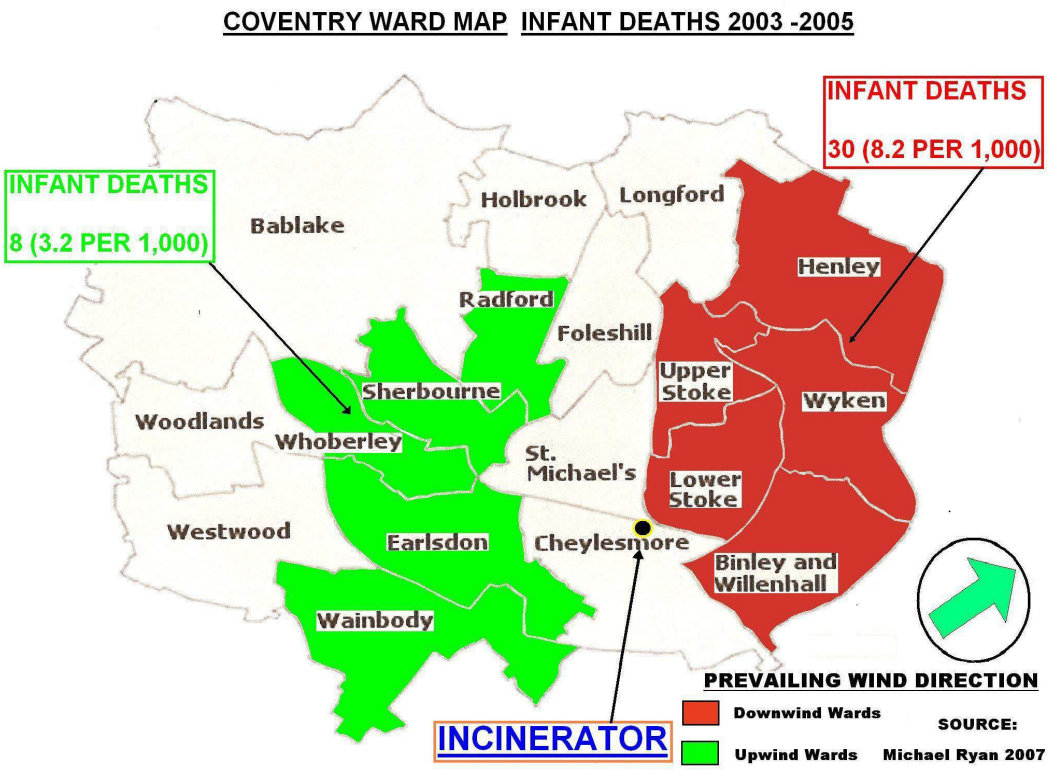
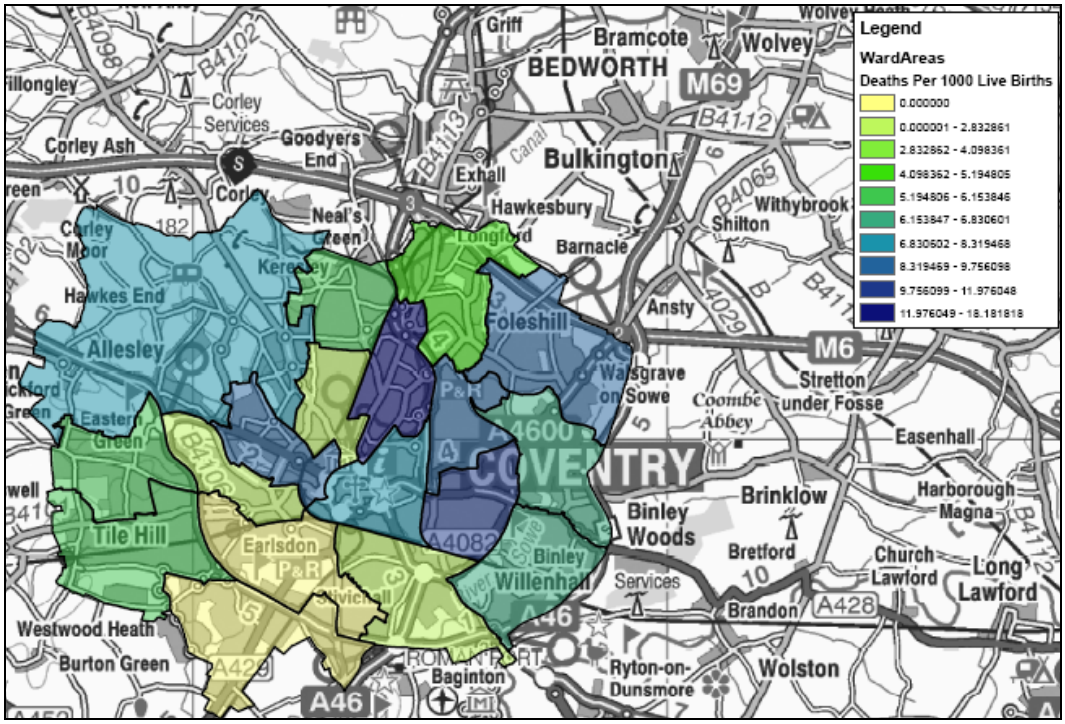


Figure 5.3.2 Infant mortality rates in the vicinity of the Coventry waste incinerator (by ward)



The information in Figure 5.3.1 indicates that infant mortality rates could be higher in the area downwind of the prevailing wind direction of the Coventry waste incinerator than in the area upwind. It does not show that $PM_{2.5}$ emissions from the waste incinerator are the cause of this difference. Indeed, other sources of ultrafine particulate matter have a more significant impact on public exposure. This suggests that the incinerator is unlikely to be the cause of the difference in infant mortality rates between the red and green areas on Figure 5.3.1.

The data in Figure 5.3.2 give a more fundamental insight into the relationship between the presence of waste incineration and infant mortality rates. The variability in infant mortality rates indicates that there are many factors influencing infant mortality. Looking at the data by ward makes it very difficult to distinguish any consistent pattern in infant mortality rates.

Conclusions

The best form of public response to the serious criticisms levelled at waste incineration facilities is in simple, straightforward terms. Ideally, each criticism could be addressed with an equally straightforward response. However, detailed scientific evaluation is needed to underpin these simple 'one liner' responses. For example, more detailed investigation indicates that there is no strong basis to recent claims that municipal waste incinerators have a significant effect on infant mortality rates. However, if a basis is found to any of the criticisms then the waste management industry and regulators should make an appropriate response to deal with the issues raised.

At present, these issues are being debated in public meetings and by the media. Public debates are likely to lead to entrenched positions, with emphasis on 'winners' and 'losers'. While it may be necessary to provide a response through these public forums, the most effective way to communicate a response to the criticisms of waste incineration facilities is on a one-to-one basis. It is preferable to help residents, decision-makers and other stakeholders reach their own conclusions after a measured consideration of the evidence.

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5.4 Particulate air pollution and risk of stillbirth: The UK PAMPER study, 1961–92

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**Presenter*

Background and objectives

A growing body of evidence suggests that exposure to ambient air pollutants, including particulates, can adversely affect the growth and development of the fetus (growth restriction, preterm birth, congenital anomalies; Dejmek *et al*., 2000; Leem *et al*., 2006; Parker *et al*., 2005; Ritz *et al*., 2002) and infant survival (Bobak & Leon, 1999a; Lipfert *et al*., 2000; Woodruff *et al*., 2006). A recent systematic review of the effect of particulate matter (PM) exposure on fetal outcomes concluded that the currently available evidence was suggestive of a small adverse effect of air pollution on fetal growth and duration of pregnancy (Glinianaia *et al*., 2004).

Previous research into risk of stillbirth associated with particulate air pollution is limited. Of the three studies included in the systematic review, all reported little evidence of an association between exposure to particulate matter and stillbirth risk (Bobak & Leon, 1999b; Pereira *et al*., 1998; Sakai, 1984). However, these studies were ecological or time-series in design with their known potential for bias. The systematic review concluded that the current evidence was insufficient to evaluate a potential association between particulate air pollution and stillbirth risk (Glinianaia *et al.*, 2004).

The aim of this study was to investigate further the potential association between ambient particulate matter and stillbirth risk, using individual level information from the UK PAMPER (Particulate Matter and Perinatal Events Research) birth database, an historical cohort study of all singleton births in Newcastle upon Tyne during 1961–92.

Study description

Study sample

The PAMPER birth cohort consists of all singletons born during 1961–92 to mothers resident in the city of Newcastle upon Tyne in northern England (current population approximately 260 000). A computer database of birth records was constructed using data from several sources, including paper-based neonatal records from the two major maternity hospitals in Newcastle. Neonatal records from both hospitals contained data on important maternal and fetal/infant characteristics, including birth outcomes used in these analyses, that is vital status at birth and gestational age. Data from birth ledgers covering the period 1961–1973 were also included to establish the full denominator for the early period of the study. However, gestational age was not available from birth ledgers and home births were thus excluded from this particular study as it was not possible to assign an exposure estimate without gestational age information.

The birth data were linked to information on stillbirths from the Office for National Statistics (ONS) for 1961–92. Among the total of 1248 eligible (resident within the study area and born in either of the two Newcastle maternity hospitals, including home births for the 1960s and multiple births) stillbirths provided by the ONS for the study period, 1222 (97.9%) were matched to the PAMPER database.

For consistency across the study period we have defined a stillbirth as the birth of a dead baby at 28 or more completed weeks of gestation (the legal cut-off in gestational age for stillbirth was changed to 24 weeks in October 1992 in England and Wales). Stillbirths with birth weight less than 500g were excluded if gestational age was unknown.

All births were assigned unique spatial identifiers (postcodes and/or grid references). For births between 1961 and 1970 (prior to the introduction of postcodes), the address at birth was assigned a postcode from a 1991 postcode book or a grid reference. Townsend deprivation score, an area-based measure of socio-economic status, was calculated at the enumeration-district level using data from the 1971, 1981 and 1991 UK census surveys.

Exposure assessment

Weekly black smoke levels were obtained from routine data recorded at 20 air pollution monitoring stations within Newcastle upon Tyne's city boundary between October 1961 and December 1992 and available from the UK Air Quality Archive. Over the whole study period, the number of monitors active in the study area during any given week varied between three and ten. Black smoke levels for each individual birth were estimated using a combination of monitored air pollution and pollution source data, date of birth, estimated date of conception (based on date of last menstrual period) and the mother's residential postcode, which identified the location at which black smoke levels were to be estimated.

The modelling process to estimate individual exposure estimates is described in detail elsewhere (Fanshawe *et al.*, 2007) and was presented at the Tenth Annual UK Review Meeting on Outdoor and Indoor Air Pollution in 2007 (IEH, 2007). Briefly, a two-stage modelling strategy was employed. First, a seasonally varying temporal trend in black smoke exposures was estimated using a dynamic linear model. Second, the remaining spatio-temporal variation was accounted for using temporal and/or spatial covariates (number of chimneys within 500 m of monitor, distance of monitor to nearest industry, type of land use and implementation of the Clean Air Act). The residual spatio-temporal correlation remaining after this process was negligible. Mean weekly exposures were estimated for each birth for each trimester of pregnancy and for the whole pregnancy period.

Statistical analysis

Potential association between estimates of black smoke and risk of stillbirth was estimated using multivariable logistic regression, adjusting for potential confounding factors. Odds ratios (OR) and 95% confidence intervals (95% CI) are reported. Non-linearity of associations between risk factors for stillbirth, in particular year of birth and black smoke, were assessed and modelled using fractional polynomials (Royston *et al*., 1999). Although variables are presented categorically in descriptive tables, all logistic regression analyses used continuous, or for parity, ordinal, data for all variables other than sex.

The statistical software package Stata, Version 9 (StataCorp, College Station, TX) was used.

Results

The PAMPER database contains details of 109 086 births, including 90 537 births with complete gestational age and residential address information, of which 812 were stillborn. Using a linear term for black smoke exposure, an unadjusted odds ratio of 1.04 per each 10 $\mu g/m^3$ (95% CI: 1.035–1.051) was seen for weekly black smoke exposure averaged across the whole pregnancy period. The unadjusted model also showed significant associations ($p < 0.0001$) between mean weekly black smoke exposure and stillbirth risk for all trimesters of pregnancy: OR per 10 $\mu g/m^3$ 1.030 (95% CI: 1.024–1.036), 1.032 (95% CI: 1.025–1.038) and 1.032 (95% CI: 1.026–1.039) for the first, second and third trimesters respectively.

However, this association was shown to be significantly non-linear. Using fractional polynomials and adjusting for year of birth, parity, sex and Townsend deprivation score, the increased risk with increasing black smoke exposure during pregnancy remained.

Conclusions and discussion

This large study over a 30-year period has shown an association between black smoke exposure during pregnancy and risk of stillbirth, with significant increasing risks with increasing exposure for all trimesters as well as the whole pregnancy period. These associations remained after adjusting for potential confounding factors, including year of birth and Townsend deprivation score, adjustment for which may over-adjust the model due to the structural correlation between both year of birth and deprivation and black smoke pollution. The effect of year of birth was also modelled using fractional polynomials, but this did not affect the significance of black smoke exposure on stillbirth risk.

If the association found in this study is causal, this would be of particular relevance to parts of the world now experiencing the levels of black smoke seen in Newcastle early in the study period. However, as this is the first study to demonstrate such an association, further research is required to confirm or refute our findings and if confirmed to identify the biological mechanisms involved.

Acknowledgements

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5.5 Discussion

Dr Metassan was asked if, for the demonstrated influence of the presence of Fenton reactions on clot structure, the concentrations of particles studied *in vitro* were comparable to the levels that are found in human blood under actual (realistic) exposure scenarios. In response, it was noted that there was a lack of information on the concentrations of particles in blood although work was ongoing in patients that might provide this information. Also, there was a need to confirm the fate of the labelled moiety in the materials studied, as instances had been identified in the literature where behaviour was not as expected. It was also noted that there was as yet no definitive evidence of any direct interactions occurring between particles and fibrin molecules. Particles had been visually located on fibrin but did not appear to show any obvious interaction.

Regarding the study on lung function in Nepalese adults, in response to a question Professor Ayres noted that the difference in altitude between the populations studied appeared to make little difference to their response to pollutants. However, a potentially important difference between the urban and rural populations had been identified: compared with urban populations, infant/childhood mortality rates in rural areas were very high, making the rural adult population studied effectively a 'survivor' population. The possibility of studying migrants between the urban and rural communities was suggested, however it was noted that migration tends to be from the rural to the urban environment and migration to rural locations is very rare. Professor Ayres also stressed the preliminary nature of some of the analyses reported, since the data had been subjected only to univariate statistical analysis to date. However, these newly established cohorts do offer the prospect of being able to extend the research to address other important issues, for example through consideration of early life events and dietary interactions. Given the potential dangers highlighted by Professor Brunekreef in relation to exposure to dust storms, a question was posed whether Nepal was subject to such events and if this might allow study of its impact on lung function. However, while 'dust days' were noted to occur, Professor Ayres did not consider that they equated to 'dust storm' conditions.

During discussion of the presentation given by Dr Broomfield it was noted that our understanding of the linkage between socioeconomic factors and mortality in populations around cement kiln sites is limited. There were also calls from some participants for increased efforts by the kiln operators – and potentially by regulators – to improve the level of monitoring. Attention was also drawn to the problems implicit in the successful communication of an understanding of the low degree of risk faced by a population living in the vicinity of a potential source of pollution, such as a cement kiln, in order to calm the fears of the local community. It was suggested that this would require the communication of complex data to demonstrate that any risk was very low; this is likely to require the difficult extrapolation of findings from UK population level data to statements that explain the significance to the local (small) community in question. While this may well be an achievable objective for much of the local population, there may always be a small percentage of the population – that may comprise a vocal pressure group – who choose not to accept either the science base or the robustness of the peer-review process, and seek to achieve a zero hazard situation rather than accepting the adoption of a risk-based approach. It was noted that, in order to achieve the best possible outcome in such scenarios, it is essential that trust and credibility are built and that any attempt at selective citation of data is rejected.

Several potential issues were raised with regard to the presentation by Dr Glinianaia in relation to the nature of the analysis adjustments undertaken and the use of chimneys as markers of a range of other risk factors. It was however noted that, with regard to the question of risk factor identification, some incomplete data are available that might clarify the situation. It was also queried whether the historic metric of soot exposure used in the Newcastle study might be a surrogate for exposure to other agents, such as PAHs, and if so, might these compounds still pose a risk to the population? Dr Glinianaia noted that there were varying amounts of data available which might allow such concerns to be addressed.

6 Epidemiology, Public Health and Policy Development

This session, chaired by Professor Klea Katsouyanni of the University of Athens, comprised five papers considering the relationship between epidemiology, public health and policy for air pollution.

6.1 Links between urban ambient particulate matter and health: Time series analysis of particle metrics

Richard Atkinson[1]*, G Fuller[2], HR Anderson[1], R Harrison[3], A Allen[3] & B Armstrong[4]

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****Presenter***

This presentation discussed the outcome of an epidemiological (time-series) study for the period January 2000 to 31 December 2005, to investigate the association between particle exposure (expressed as a range of different metrics) and daily mortality and emergency hospital admissions in the North Kensington area of London. This included consideration of all causes, cardiovascular and respiratory mortality data. However, at the request of the author no detailed abstract is available for inclusion in this report.

6.2 Quantification for health impact assessments: Linking deaths, lives and values

Brian Miller, Institute of Occupational Medicine, Edinburgh

This meeting reports the end of a three-year programme of support for the development at Institute of Occupational Medicine (IOM) of life table methodology in health impact assessment, funded by the Department of Health and latterly administered by the Health Protection Agency.

Central to the methodology is the fact that survival patterns and life expectancy are functionally linked to age-specific mortality rates or 'hazard rates'; standard actuarial life table methods are used to construct survival functions from hazard rates using simple arithmetic. Hazard rates are usually estimated as the ratio of observed age-specific deaths to the mid-year population at that age.

$$\hat{h}_i = \frac{d_i}{m_i}$$

Survival from one birthday to the next is then estimated as

$$\hat{s}_{i+1} = \frac{2-\hat{h}_i}{2+\hat{h}_i} = 1 - \frac{2\hat{h}_i}{2+\hat{h}_i}$$

and the probability of survival to a given k^{th} birthday, F_k, is estimated as the product of these.

$$\hat{F}_k = \prod_{i=0}^{k-1} \hat{s}_{i+1}$$

Such calculations, if based on current hazard rates, make the strong assumption that future hazard rates will be the same as current. However, the calculations can be applied to any set of hazard rates, which allows assumptions about the behaviour of future rates other than that they will remain constant.

In particular, this allows the comparison of life expectancies between two scenarios with different hazard rates, for example if we assume that reductions in ambient particulate pollution will cause a corresponding reduction in mortality rates. We have shown that a 1% reduction in all-cause mortality hazard rates in adults aged 30+ produces a gain in life expectancy of about 10% of a year, and that gains for other small amounts of change in the hazards are close to proportional.

The population affected by a change in pollution levels will be distributed across all the ages, but we can perform similar calculations and comparisons across the remaining lives of each age-specific sub-population, and on future birth cohorts, and combine the results in a wide variety of summary combinations (Miller & Hurley, 2003). These methods have been developed at the IOM in a suite of Excel spreadsheets named IOMLIFET, which is distributed free of charge to interested users. The methods have been adopted and used by a number of organisations and multinational projects.

Attaching monetary values to changes in mortality risks for cost–benefit analyses is problematic. A widely used method calculates differences in numbers of deaths and applies to these a value of statistical life (VOSL). Based on willingness-to-pay surveys, this is usually a single value regardless

of the age at which the death occurs, so it ignores the amount of expected remaining life. Perhaps more importantly, applications usually ignore the fact that changes in death rates have a knock-on effect on the sizes and age distributions of populations in future years. The concepts of 'lives saved' or 'deaths avoided' often used in reporting is also a misnomer since everyone in a population dies eventually, and altering the hazard rates can only change the temporal pattern of deaths (Brunekreef *et al*, 2007), in a way that can be demonstrated and quantified using IOMLIFET.

An alternative route to valuation would be to attach a value to a year of life. This could be a single value, or could differ by age. In fact, since the results of the IOMLIFET calculations allow access to differences in life-years by both age and calendar year, any scheme of weighting can be imposed on the life-years, and this can be extended to other weights such as future monetary discounting, QALYs and DALYs, etc.

Communicating the results of such calculations has not always been easy. Newspapers and other mass media find that quantifications of lives saved or unnecessary deaths make good headlines, and often ignore any qualifications on the statements (if these are reported at all). By comparison, average gains in life expectancy seem less compelling. If we assume that a 10 $\mu g.m^{-3}$ reduction in $PM_{2.5}$ leads to a 6% reduction in all-cause mortality hazard rates, and apply this change to recent hazard rates for England and Wales, we predict an average gain in life expectancy of around 7 months. Over a whole population, this adds up to a huge saving, but it may not seem a lot per individual, and such findings have had demonstrably less media appeal than when expressed as so many thousand lives 'saved'.

Other suggestions for expressing changes in mortality include Brenner's (1993) 'rate advancement period', or the 'real age' concept now appearing on health oriented websites. However, mortality rate advancements or shifts in 'real age' of a few months on average seem still to lack media punch.

An alternative way of risk communication is to place the risks in context with other, hopefully better understood, risks. We have compared the effects of a 6% reduction in all-cause mortality hazard rates, to mimic a 10 $\mu g.m^{-3}$ reduction in $PM_{2.5}$, with the effects of eliminating deaths from motor vehicle transport accidents (MVTA) and of eliminating second-hand tobacco smoke (before the introduction of smoke-free legislation). These calculations, based on cause-specific impact assumptions, suggested that the gains from reduced air pollution would be similar between the sexes, in males around three times the effects of eliminating either second-hand smoke exposure or all MVTA, and in females nearer seven times the impact of MVTAs (Miller & Hurley, 2006).

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6.3 Modelling solvent dispersion, chemistry and health impacts to inform policy development on VOC control

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**Presenter*

Fate modelling of VOCs to inform the development of the National Emission Ceilings Directive and the Gothenburg Protocol has focused on their impacts via the formation of ozone, with limited attention paid to wider impacts or the variation in the properties of different species in the overall VOC mix. Against this background, Defra has for a number of years funded the development of the Master Chemical Mechanism (MCM), which seeks to describe the fate of VOCs emitted to the atmosphere. While much of the research on the MCM has focused on tropospheric ozone formation its potential is considerably broader, with links also to formation of secondary organic aerosols (SOA) and to climate change. Add in the fact that many VOCs are also directly harmful to health, and it is clear that the problem of VOC control has many different facets, and that policy based on individual concerns may not provide the all-round best solution to the problems posed by VOC emissions.

The work performed under this project has sought to take full advantage of the potential of the MCM to develop a far more complete framework for policy evaluation of controls on VOCs than has previously been available. To illustrate this framework a case study has been performed to consider alternative solvents to trichloroethylene, also known as trike, which has been used extensively for degreasing metals prior to coating. These options were subjected to cost-effectiveness analysis to identify which offers the most efficient alternative from a purely financial perspective. The potential impacts of alternative solvents were then reviewed to provide a semi-quantitative perspective on which may be likely to offer an advantage to the target chemical. Screening can be applied at this stage to assess whether any options clearly stand out as advantageous or disadvantageous to provide better focus for the more detailed assessment of benefits that follows.

For options that need further investigation the framework then proceeds through:

- life cycle analysis to provide a thorough understanding of inputs and outputs of materials and energy
- impact quantification (for the solvent itself via toxic effects, ozone formation, SOA formation and contribution to greenhouse gas (GHG) emissions, and for effects of other life cycle emissions that appear significant)
- economic valuation of impacts, where feasible
- multi-criteria assessment to ensure that non-monetised effects are not lost
- use of cost–benefit analysis
- uncertainty analysis to check the validity of comparisons of costs and benefits.

It is not necessary to apply the full framework in all cases. However, its application of the full framework is necessary in the context of this project to illustrate how the different methods, which are often considered to be in competition, can be used in combination to provide more thorough guidance than would be possible by using methods in isolation of each other.

This work has been funded by Defra as part of ‘Modelling of Tropospheric Ozone’, Project AQ0704.

6.4 An evaluation of the impact of the congestion charging scheme on pollution concentrations in London

R Atkinson[1]*, B Barratt[2], B Armstrong [3], HR Anderson[1], SD Beevers[2], IS Mudway[2], D Green[2], RG Derwent[4], P Wilkinson[3], C Tonne[3]& FJ Kelly[2]

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****Presenter***

This presentation discussed various changes over time that were identified in the levels of a number of air pollutants in various areas of London following the introduction, on 17 February 2003, of a congestion charging scheme that operated at peak periods of traffic movement. However, at the request of the author no detailed abstract is available for inclusion in this report.

6.5 Waste derived fuels in cement kilns, public health implications and response: Assessing the evidence

Patrick Saunders

Health Protection Agency, Didcot, Oxon

Summary

The Health Protection Agency (HPA) has produced a critical assessment of the public health impact of burning refuse-derived fuels in cement kilns.

Introduction

The HPA has reviewed the impact of using waste derived fuels (WDF) such as solvents, sewage pellets and tyres on emissions and public health.

Method

A detailed review of the published literature, together with emission data from four trials using WDF and specification data for the fuel input has been conducted and considered by the Committee on the Medical Effects of Air Pollutants (COMEAP).

Results

Portland cement is a binder formed from ground clinker made from a mixture of lime (CaO) and silica (SiO_2) with a small proportion of alumina (Al_2O_3) and iron oxide (Fe_2O_3). High gas temperatures are required especially during the sintering phase where the CaO reacts with the other components in a liquid state to form calcium silicates, aluminates and aluminoferrite, the main components of cement.

The cement process is very energy intensive and the industry has moved to reduce fuel costs including by sourcing alternative non-fossil derived fuels. The use of these WDF is now widespread, subject to tight statutory control and an agreed industry code of practice. It has major advantages for the environment, industry and international obligations. However the practice remains the source of much public concern.

The main releases to air are from the kiln stage via the kiln exhaust gases, clinker cooler exhaust and any bypass gases. Several studies have suggested that the use of WDF is no more polluting than conventional fuels, and for some key emissions less so. There are benefits to the UK in meeting obligations under the Kyoto accord and European legislation.

The Waste Incineration Directive (WID) requires the regulator to set mandatory emission limits for many of the key pollutants such as dust, HCl, NO_x, metals and dioxins. Many of the WID limits are tighter than previously set as permit conditions.

There are four main groups of WDF.

- Waste liquid fuel (WLF) or secondary liquid fuels (SLF). These are blended residues from recovered or recycled industrial waste solvents and waste solvents/hydrocarbons that cannot be recycled. This group also includes recovered fuel oil (RFO) derived from used or discarded oil from automotive, shipping and industrial lubrication applications.

- Tyres, which are either whole or chipped and have the same high calorific value as coal. The steel reinforcing from the tyres oxidises in the kiln and replaces a portion of the iron that would otherwise be added to the raw material mix used in cement manufacture.
- Solid recovered fuels (SRF), which include refuse-derived fuel (RDF) and mechanically and biologically treated municipal waste (MBT) such as Profuel® and Climafuel®. This group covers a wide range of waste materials including residues from municipal solid waste and industrial or trade waste which has been processed to recover metal, glass and plastics and remove stones and other non-combustibles.
- Biofuels, such as meat and bonemeal (MBM) and processed sewage pellets (PSP). MBM is produced at animal rendering plants during the high-temperature processing of animal remains comprising mainly abattoir waste. It is a granular solid residue left after extracting fat (tallow) during the rendering process. PSP is made from the sludge from sewage works after treatment of the sludge by drying and heat treatment to produce a sterile, dark glassy pelletised material.

The nature of the cement kiln process means that many potential pollutants are destroyed or trapped in the clinker. The very high temperatures and long residence times produce a highly efficient environment for the destruction of organic compounds including dioxins and furans (>99.99% efficiency) and the highly alkaline conditions decompose chlorinated organic wastes and acid gases. Counter-current flow of feed material and combustion air means many of the pollutants which potentially could be released to air are trapped in the clinker. Heavy metals are absorbed by the clinker due to the high alkaline content of the clinker and the scrubbing action within kilns. Refractory metals such as Ba, Be and Cr tend to be incorporated into the clinker at approximately 99.9%, and 99.5% of the semi-volatile metals such as Cd, Pb, Zn are also trapped in the clinker.

Particulates are removed and returned to the clinker. The industry has traditionally used electrostatic precipitators but has increasingly moved to fabric bag filters enabling some works to reduce dust emissions to single figures (emission limit value is 30 mg/m^3).

The few studies specifically examining public health and use of WDF in cement kilns are reassuring. In addition a great deal is known about the process characteristics, and the HPA has assessed the fuel specification data and the results of four trials with experts from the academic communities.

Conclusion

The use of WDF in cement kilns, effectively operated, monitored and regulated in compliance with good practice, is no more hazardous than using conventional fossil fuels.

6.6 Discussion

The influence of spatial location on exposure was queried in relation to the time-series study presented by Dr Atkinson, and it was noted that there was some evidence for such effects. It was also noted that, in order to better interpret the findings of the study within a toxicological context, it would be necessary to present data in terms of effect per unit increase in concentration rather than using a quartile basis as the metric for exposure in the analysis. The second study presented by Dr Atkinson, on the changes following the introduction of the congestion charging scheme in London, had highlighted some interesting, indeed unexpected, findings. For example, there was some evidence that the type of traffic passing within the scheme boundary and surrounding areas had changed, with the willingness of 'high polluting' vehicles to enter the area possibly being reduced to a greater than expected extent. There remained issues concerning study interpretation, particularly possible interrelationships/interactions between various pollutants. For example, neither the relationship between NO, NO_2 and O_3 distributions across the study areas, nor the influence of changes in public transport provision, were clearly understood. Such aspects were the subject of ongoing analyses, and it may be that some of the effects detected are chance findings (which would not be surprising given the large number of measures and outcomes considered). It was hoped that additional analysis would allow examination of consistency of effects across analyses. A note of caution was raised by the researcher who noted that, although the study had investigated a wide range of factors, it was not designed to enable effects to be attributable to specific individual factors (i.e. factors were not necessarily mutually exclusive and may interact in various ways). It was also noted that, while model development was an initial intention of the project, problems relating to the lack of any control data *per se*, data smoothing and uncertainty issues made the development of robust models very difficult in practice. Also, it was stressed that the original intention for introducing congestion charging was not to reduce air pollution, but to cut traffic congestion, so any changes in pollution levels should be regarded as a secondary consequence, not as evidence of success of the scheme. In fact, Transport for London did report an initial marked reduction (30%) in congestion and a 17% fall in traffic volume – which was taken by many as a sign of success. However, these do not seem to have been maintained and it now appears that, while overall volume may still be suppressed, congestion is again increasing. It was also noted that, while limited in nature, available information on the impact of introducing the low-emission zone in London suggests that the magnitude of reduction in pollution has been small, indicating that introduction of a single measure of this type is likely to be insufficient to make a significant difference in the longer term. The findings from the study on quantification of health impacts presented by Dr Miller are already bringing benefits, with the use of the output aiding development of future policy in this field.

In relation to the presentation by Dr Saunders, it was noted that evidence exists on the value of using kilns as a way of eliminating waste compared with alternative strategies. It was however agreed that, on a site-specific basis, there is often a problem with regard to availability of robust *ad hoc* and *post hoc* exposure information, and discussions with the public may be clouded because of concerns regarding a range of other issues relating to the operation of a kiln facility in the area. There was concern voiced from some quarters that regulators base decisions on a small number of highly controlled trials, while there is a history of problems with regard to the general quality of ongoing facility operations and performance. It was agreed that this can at least be perceived as an issue by the public. However, Dr Saunders suggested that this aspect is really a matter for the regulating authorities to ensure discharge consent requirements are met, rather than an implicit problem with the derivation of the consent requirements by the HPA (developed with input from COMEAP). Nonetheless, there is a need for regulators to ensure that adequate monitoring and controls continue. Another participant supported this view noting that, from experiences gained in the study of one particular kiln facility, the plant was operated well and that any risk to the public in the area would have been exceedingly low, while in another instance it was found that there was an operational problem in the plant that had led to a pollution episode. This had been the subject of a legal case and resulted in a large fine. It is thus apparent that society should seek to control poorly operated plants and not ban activities based solely on the basis of 'classes' of plant or industry.

7 Parallel Discussion Sessions

Two parallel discussion sessions were held to consider the topical subject areas of particle toxicology and the use of distributed lags in epidemiological research. Each session was introduced by an appointed facilitator, who briefly outlined the key issues for discussion.

The main points addressed by session participants are summarised in the sections below, suggesting research questions in need of further evaluation.

7.1 Particle toxicology

Facilitator: Annette Peters

Rapporteur: Paul Harrison

A short presentation by the facilitator, Dr Annette Peters, focused subsequent discussions on the main underlying factors thought to influence the toxicity of particles. Three key metrics were identified: the physicochemical properties of the particles; the physiological and toxicological response by tissues/organs (the 'compartments') that are most likely to be exposed to the particles; and the susceptibility of the host to particle–compartment (tissue, organ) interactions.

The importance of particle properties was addressed first, drawing on presentations made at the meeting that showed a correlation between increasing particle numbers, rather than particle mass and rises in cardiovascular events. Early work on the health effects of air pollution indicated that it was particle mass that appeared to represent the greatest hazard to health. However, as mechanisms could not be determined and as effects of very small mass-doses seemed unlikely, it was proposed that mass may be a surrogate for particle number, for which mechanisms of effect could be more easily suggested. More recently, it has been shown that particle number is not closely correlated with particle mass, and it is necessary to consider both number and mass aspects when evaluating health effects. For nanoparticles, particle number may be the more appropriate parameter. Furthermore, many particles are produced by vehicular traffic, particularly ultrafine particles, and many of these are not solid but are in liquid state. These have an as yet unknown mechanism of effect in the airways and lung tissue despite having being associated with adverse effects on health.

Particle size is an important consideration for deposition in the pulmonary compartments and duration of epithelial contact (Table 7.1.1). While particle sizes determine their fate in the body, the particle surfaces are likely to be the key to their toxicological action. Solubility and other physicochemical properties of particles, including particle composition, affect the behaviour of particles in the lung and the likelihood of entry into the blood. The soluble constituents of larger particles deposited in the conducting airways might be absorbed into the blood stream. Only a small proportion of most inhaled particles can be expected to enter the bloodstream. However, nanoparticles have been detected in the blood but toxic effects are not yet clear. In fact, the use of nanoparticles for medical diagnostic purposes suggests an absence of toxic effect. More research is needed to understand the fate and behaviour of nanoparticles in the body, particularly in the blood stream, as presence need not lead to negative effects (damage). Conversely, diffusion of particles across the epithelium may trigger a cascade of release of inflammatory mediators – with direct and indirect effects.

The study of ozone, which, like particles, has the potential to cause an effect at even low doses, may provide insights into the possible health effects of particles. It was suggested that the physiological/toxicological responses resulting from exposure to ozone are analogous to the effects of nanoparticles – such as inflammatory and immune system responses resulting in the activation of macrophages (Table 7.1.1). Autonomic responses may also result, but there is very little information

concerning the mechanism of activity. Contaminants carried by nanoparticles, such as polycyclic aromatic hydrocarbons, may also exert an effect on health. Transfer of such materials to the fetus was seen as potentially important. It is known that overload of macrophages occurs at lower volume doses of nanoparticles than is the case for larger particles, but the likelihood of this effect occurring at ambient concentrations is low. In lung tissue, macrophages do not respond to nanoparticles or non-clumping material and such particles are more likely to result in oxidative stress, particularly in the alveolar areas. Pulmonary tissue is continually exposed to free radicals and hence oxidative stress, but there are no comparisons of 'normal' free radical exposure to the increased levels of free radicals caused by pollutants. Whilst smoking increases the numbers of free radicals in lung tissue, there is no indication that levels achieve a tipping point, suggesting that lung tissue is able to withstand elevated oxidative stress. The placenta, which consists of both the maternal and fetal endothelial systems, is particularly sensitive to oxidative stress but there has been very little work looking at effects on the placenta and thence the fetus. Effects on the autoimmune system, should they occur, may also have consequences for heart function and heart rate variability.

Table 7.1.1 Particle–compartment interaction

Compartment	Particle	Particle properties	Pathophysiology	Metric (%)*
Airways	Course + fine + ultra-fine	Oxidative stress, water soluble	Activation of autonomic nervous system	10–30 PM
Alveolar	Fine + ultra-fine	Solubility, oxidative stress, metals, surfaces + interactions on surfaces	Inflammatory and immune responses. Depletion of antioxidant defences?	70–90 PM
Epithelium	Solid ultra-fine + components	Surface properties, oxidative stress	†	<1 PN
Blood	Solid ultra-fine + components	Surface properties, oxidative stress	†	<1 PN
Other organs/fetus	Solid ultra-fine + components	Surface properties, oxidative stress	†	<1 PN

*PM, particulate matter; PN particle number; † not considered in this discussion

The area of pulmonary tissue affected by particulate matter is likely to be localised compared with, for example, the more uniform exposure expected for a gaseous substance such as ozone. It can therefore be expected that oxidative stress resulting from particulate matter is exerted in restricted areas of the lung causing localised inflammatory responses or antioxidant responses. Events in the lung also trigger bone marrow effects. While ozone, which is rather insoluble, is able to penetrate deep in the lung, the deposition of particles in the alveoli is highly dependent on size, with peak deposition occurring at diameters of 20–30 nm. Greater understanding of this behaviour is required and of how the particles interact with epithelial cells and lung surfactant. It was suggested that ozone could be used as a control for further studies on the toxicological effects of particles due to the comparatively high level of understanding of ozone toxicology. For instance, data exist on the relationship between ozone and surfactant that reveal that there is a sufficient amount of surfactant to maintain lung function at high levels of ozone. However, a key location for nanoparticles eliciting effects could be the interstitium rather than the alveolar space. Surfactants vary in type and concentration between humans and animals, making read-across from animal data possibly misleading. This may also be the case between smokers and non-smokers.

Five potential compartments were suggested as being the main particle interaction points (Table 7.1.1).

Summary of key points

The output from this parallel discussion session can thus be summarised as follows.

- Particulate mass as a metric is not a surrogate for particulate number; mass and number may have different 'targets' and consequences. Hence consideration still needs to be given to particle surfaces as an unmeasured but critical determinant of particle toxicity.
- The behaviour of liquid particles, and if/how they differ from solid particles, requires further work.
- Experimental studies are required to compare the effects of ozone and particles.
- There is a need to identify the location where particles exert their effects on the respiratory system.
- Greater understanding is required of the processes mediating ultrafine particle–immune system interactions.
- Clarification of the factors influencing lung permeability to various particle types is needed.
- The mechanisms by which particles impact on health endpoints via interaction with the autonomic system should be a focus of future work.
- Other tissues (particularly the placenta) require evaluation as organs that may be susceptible to toxicological insult (e.g. through oxidative stress mediated mechanisms).
- Dosimetry and the effects of particular particulate size ranges should be considered.
- Susceptible groups need to be identified.

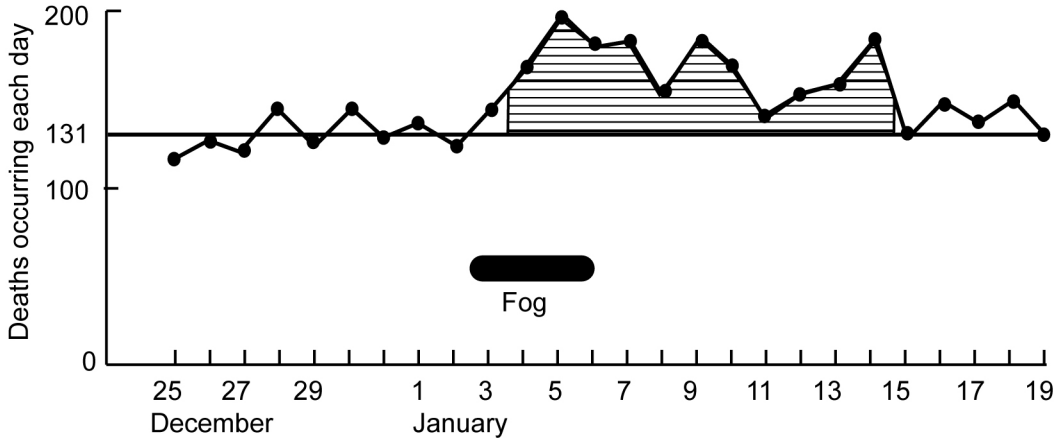
7.2 Distributed lags in epidemiology: Should we be using distributed lags for quantification?

Facilitator: Ross Anderson

Rapporteur: Heather Walton

Professor Anderson opened discussions by outlining the historic application of distributed lags in the investigation of deaths following pollution episodes, perhaps the most famous example being the 1955/56 London fog episode (see Figure 7.2.1).

Figure 7.2.1 Deaths (all causes) occurring each day in London Administrative County from December 25, 1955, to January 19, 1956



Source: Logan (1956)

The shaded area represents deaths in excess of 131 per day during period January 4 to 14

Over subsequent years, further studies on other pollution episodes and the introduction of new statistical techniques have resulted in major shifts in the purposes to which this type of study has been put. There is increasingly a move towards the use of such studies to inform on the nature and size (magnitude) of any aetiological associations identified between an airborne pollutant and health effect, a change that has been driven in part by the needs of regulators to develop policy. This process has led to new understandings of the potential for larger effect sizes for distributed lags, the length of life lost for the deaths that may be picked up in time-series studies (i.e. more than a few days), and apparent differences in the lag patterns for different pollutants in relation to various health outcomes. However, this topic was thought to remain a contentious and poorly understood aspect of epidemiology, highlighting the need to consider a number of basic questions.

- What are distributed lags?
- Should distributed lags rather than single lags be used for quantification?
- To what extent is variation in effects at different lags due to random variation and to what extent is it due to biological mechanisms?
- Should we be meta-analysing lag structures to check if there is consistent information on the importance of different lags in time-series studies?
- What/how do distributed lag models tell us about harvesting?

Dr Richard Atkinson of St George's, University of London, then presented examples from published work on the insights that can be gained through application of both systematic review and meta-analysis, which highlighted that the interpretation of data may be significantly affected by decisions on what lag or combination of lag periods are to be used to best characterise an association. Dr Ben Armstrong, London School of Hygiene and Tropical Medicine, subsequently considered, in turn, the questions posed to the group.

It was noted that analyses could be performed in two ways: looking forward from the time of exposure to the effects that subsequently occur at various time points, or looking back in time from when the effect(s) are noted to the changes in pollution levels at various time points in the past. In practice, the researcher is generally faced with a more complex situation in which the exposure event occurs over a number of days and changes are seen in the endpoint metric also over a number of days, possibly overlapping with the latter stages of the exposure episode. This can be further complicated by different pollutants showing different lag responses for the same endpoint. In such instances, complex addition methods are required. It was also noted that there is evidence that the inclusion of longer lag periods may result in larger effect estimates (Table 7.2.1).

Table 7.2.1 Example of variability of effect estimates depending on model used

Source	Metric	Estimated effect of PM_{10} (per 10 µg/m^3 increase) on indicated endpoint			
		Percent		95% CI	
	Time series				
Schwartz (2001)	All daily deaths	0.89%		0.61–1.16	
		Cardiovascular disease		Respiratory disease	
Zanobetti *et al.* (2003)	Deaths from indicated cause*	Percent	95% CI	Percent	95% CI
	Mean lag 01	0.69	0.3–1.08	0.74	-0.17–1.66
	Distributed lag:				
	Fourth degree	1.99	1.44–2.54	4.21	1.70–6.79
	Unrestricted	1.97	1.38–2.55	4.20	1.08–7.42

Combined data for 10 European cities, unconstrained distributed lag and fourth degree polynomial distributed lag models for period up to 40 days, using bootstrap methods

Adapted from Schwartz (2001) and Zanobetti *et al.* (2003)

Fitting distributed lags may be achieved through application of multiple regression analysis. In this, the statistician sometimes seeks to limit variation through constraining the beta coefficients for each day to be equal. This is arithmetically equivalent to fitting a model using the average of the pollution days. Some patterns can be due to the constraints but it was suggested that such techniques have been shown not to overly influence the *total* magnitude of any effect determined. This supports the use of more robust approaches wherever possible. Thus, in response to the basic question: "Should distributed lags rather than single lags be used for quantification?" the answer: "Yes, if available" was considered reasonable.

When considering the extent to which variation in effects at different lags are due to random variation and to what extent they may reflect underlying biological mechanisms, it was noted that a number of issues exist. One such issue relates to the retrospective selection of the lag over which the effect is considered to occur, as this may lead to *post-hoc* bias. Use of *a priori* selection of the lag period can, however, lead to problems if the period chosen does not reflect the actual lag periods occurring in

practice (e.g. *a priori* selection of a one-day lag, but effect actually experienced after a two-day lag). Use of distributed lag analysis avoids these problems, although there may be a loss of precision if unconstrained distributed lags with a large number of variables are used. Such problems were suggested as being addressable through drawing on information available either *a priori* or generated within the study (used to inform on lag period selection) and through careful use of distributed lag models. (Some suggested choosing lag 0–1 mean as a compromise.) Application of meta-analysis techniques to study the lag structures was, however, strongly recommended as this allows one to check whether there is consistent information on the importance of different lags in time-series studies. Also, such approaches may inform on mechanisms and reduce choice problems. It was noted, though, that a drawback of such techniques is their reliance on a high level of statistical expertise (an example was given of the benefits of using non-linear distributed lag models to investigate the relationship between temperature and cardiovascular disease).

With regard to the study of so-called harvesting effects, it was noted that theoretically there should be a compensatory fall following a harvesting episode due to pollution, but there is a danger that this could be masked by the application of smoothing techniques, and the extent of such masking would be influenced by the degrees of freedom applied to the polynomial. Visual inspection of the results was recommended before modelling. It was queried if there have been any attempts to apply Bayesian techniques to the study of distributed lags, but participants were not aware of any examples.

There was some discussion on what length of lag should be considered in order to identify whether the effect seen actually represented a harvesting situation. It was suggested that if there was no evidence of a subsequent compensatory fall in the level of the endpoint being considered after approximately 40 days, then it would be reasonable to conclude that the change seen did not represent harvesting. Others drew attention to potential problems in being able to interpret the data because of the possibility that there may be repeated pulses of pollutant exposure over such a length of time, and this was noted as a particular problem if many variables (pollutants) were being considered in the study. The possibility of undertaking animal studies using various exposure regimens to provide data on which to base investigation of the statistical analysis of multiple pulses was briefly discussed. It was also pointed out that harvesting was not just a statistical issue, since there was an outstanding need – at the level of policy makers – to better define what should be considered as 'harvesting' and the lengths of time over which such effects would be considered important (days vs weeks vs months, etc). It was noted that both regulators and journalists tend to focus simply on the term 'deaths brought forward', but this may not be either helpful or meaningful. The potential benefit to policy development of the more robust and informative statistical techniques was noted. However, it was noted that there are still considerable national differences in the preferences of government regulators.

Summary of key points

When reporting back to plenary, Dr Walton identified several key conclusions.

- The use of distributed lags will result in detection of a greater effect size than single lag models, and it is important that the significance of this difference is better understood.
- Distributed lags allow comparison of exposure events and their effects over several days (and the time periods need to be combined to order to estimate the magnitude of the total effect).
- Distributed lags are useful in quantification, and may allow one to infer some biological plausibility of the effects seen.
- There are important issues remaining with regard to the extent of 'harvesting' that may occur as a result of air pollution.
- The use of distributed lags is preferable for quantification, but it is important not to ignore the single lag time series evidence as this constitutes most of the overall evidence.

- There is a need to carefully define the nature of the effect that is to be quantified, and to ensure that the data are interpreted correctly; thus, for example, the use of an appropriate time period is essential (since over a long enough period, mortality of the population will be 100%).
- Meta-analysis provides a very useful tool and, if used correctly, may inform on the nature of differences in the mechanism of effect for different health outcomes (e.g. it is possible to detect consistent differences in the lag of the effect on the cardiovascular and respiratory system, with lags being longer for respiratory effects).

In discussion, it was noted that the concentration range can be smaller for a (constrained) distributed lag (e.g. a 40-day average could be smaller than a 1-day average). This, of itself, can give a larger coefficient – the variance needs to be quoted too.

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8 Posters

In all, sixteen posters were presented over the course of the workshop. These addressed a wide range of topic areas, as might be expected given the scope of the meeting.

8.1 Vapour:particle phase distribution of hexabromocyclododecane (HBCD): Are PUF disk passive samplers suitable for monitoring HBCD in indoor air?

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Background and objectives

HBCD is a high production volume brominated flame retardant with a global demand of 16 700 tonnes in 2001. It is used as an additive to expanded and extruded polystyrene foams for thermal insulation of buildings, back-coating of fabrics and, to a lesser extent, in high-impact polystyrene (HIPS). Commercial HBCD mixtures consist mainly of the α-, β-, and γ- diastereomers with the last predominant (>75%). HBCD is considered ubiquitous in air, dust, sewage sludge, sediment, aquatic invertebrates, fish, birds, eggs, human milk and plasma samples (Covaci *et al*., 2006). While PUF disk passive air samplers are employed increasingly to monitor persistent organic pollutants in indoor air, they essentially sample only the vapour phase. Very little is known about the vapour:particle phase distribution of HBCD in indoor air as there is only one study which reported the flame retardant to be present largely in the particulate phase of outdoor air (Hoh & Hites, 2005).

The objectives of the current study are as follows.

- To determine – for the first time – the atmospheric phase distribution of HBCDs in indoor air using active air sampling
- To investigate the feasibility of employing PUF disk passive samplers for monitoring HBCDs in indoor air
- To carry out a calibration experiment to determine the passive air sampling rates for the three principal HBCD diastereomers using two passive air sampler configurations
- To compare the concentrations of HBCDs derived for indoor environments using conventional active air sampling apparatus with those obtained using the PUF disk sampling configurations for which air sampling rates were obtained

Study description

Active air sampling for determination of vapour:particle phase distribution of HBCDs in indoor air was conducted in two offices using a Graseby–Andersen Hi–Vol sampler fitted with a total suspended particulate (TSP) inlet modified to hold a standard PTFE back-coated glass-fibre filter (GFF, 0.8 mm pore size, Whatman) and a pre-cleaned polyurethane foam (PUF) plug (827 cm^3 volume).

PUF disk passive samplers' calibration was conducted in September 2007 in a temporarily vacant office microenvironment (distinct from the two offices monitored earlier, and for consistency,

identical to that employed in our earlier calibration for PBDEs and PCBs). Fully sheltered passive samplers (n = 8) were deployed over a 50 d period at a height of 150 cm with a minimum distance between samplers of 50 cm. PUF disks were harvested at 10 d intervals over the 50 d of the experiment. To ensure that detectable concentrations were provided by the passive samplers at the 10 and 20 d sampling intervals, three and two samplers were harvested and combined for analysis at these times, respectively. The analyte masses present in these combined samples were subsequently normalised to a single PUF disk equivalent mass for the purposes of the calibration. Concurrent with the deployment of the passive air samplers, a single active air sample was taken covering the full 50 d duration of the experiment. The calibration was also conducted in identical fashion simultaneously in the same room, but using 'part sheltered' PUF disk samplers with the bottom housing removed. Active air sampling for the PUF disk calibration was performed using a low-volume pump (Capex L2X) operated at a flow rate of 6 L min^{-1} to yield a single sample comprising approximately 430 m^3 air. The particulate phase was collected by employing a 47 mm membrane filter (1.0 μm pore size, Whatman) housed in a standard open-face 47 mm filter holder airside of the PUF plug sorbent. Two PUF plugs (4 cm diameter × 8 cm length) housed by a glass holder (3 cm diameter × 25 cm length), were used as a gas phase sorbent.

Samples were Soxhlet extracted, and extracts purified by washing with H_2SO_4 and Florisil chromatography prior to analysis using LC/ESI/MS/MS.

Results

In both indoor samples, the majority (~65%) of each diastereomer is present in the vapour phase (Table 8.1.1). This suggests that PUF disk passive samplers are likely to be appropriate for sampling HBCDs in indoor air.

Table 8.1.1 Concentrations (pg m^{-3}) of HBCDs in indoor air

	Concentration			
Sample id	**α–HBCD**	**β–HBCD**	**γ–HBCD**	**ΣHBCD**
Office 1 Active HiVol particle phase	16	11	53	80
Office 1 Active HiVol vapour phase	32	21	106	159
Office 1 Passive sampler	32	23	116	171
Office 2 Active HiVol particle phase	19	10	65	94
Office 2 Active HiVol vapour phase	38	20	131	189
Office 2 Passive sampler	39	22	138	199
Office 3[a] Active LoVol particle phase	30	22	69	121
Office 3 Active LoVol vapour phase	57	42	139	238

[a] Office 3 is the location in which the calibration experiment was conducted.

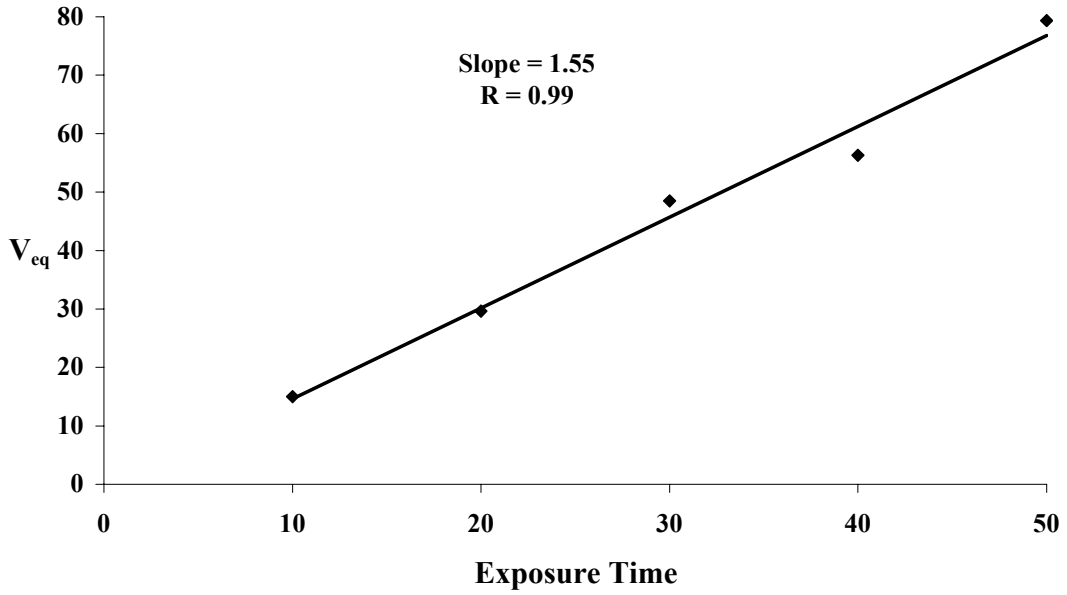
To determine the passive sampling rates of each diastereomer, the equivalent air volumes sampled by each PUF disk over a given exposure period, V_{eq} (cm^3), were calculated using Equation 1.

$$V_{eq} = \frac{M}{C_A} = k_A \; A_{PUF} \quad \Delta t \qquad (1)$$

where M is the mass of compound sequestered by the PUF disk (pg) within the deployment period, C_A is the concentration (pg cm^{-3}) of the target analyte in the air being sampled, k_A is the air-side mass transfer velocity (cm sec^{-1}), A_{PUF} is the exposed macro surface area of the PUF disk (cm^2), and Δt is the sampling period (sec). The V_{eq} values were converted to m^3 units and plotted against the exposure time of the PUF disks (d). The slope of the linear regression plots obtained (Figure 8.1.1) is defined as the passive air sampling rate (R, m^3 d^{-1}) of the PUF disk samplers for the corresponding diastereomer.

Strong correlation (R values >0.987) between values of V_{eq} and PUF disk exposure time for each diastereomer, regardless of sampler housing configuration, demonstrate linear uptake of HBCDs for the passive samplers over the 50 d calibration period. PUF disk passive sampling rates for the part-sheltered configuration were 1.38, 1.54, and 1.55 m^3 d^{-1} for α-, β-, and γ-HBCD, respectively. These were nearly double those derived for the fully sheltered configuration – 0.87, 0.89, and 0.91 m^3 d^{-1} for α-, β-, and γ-HBCD, respectively.

Figure 8.1.1 Plot of equivalent air volume (V_{eq}, m^3) versus exposure time (d) for γ-HBCD and the part-sheltered PUF disk passive sampler configuration. Slope = passive air sampling rate (m^3 d^{-1})



We also calculated air-side mass transfer coefficients (k_A) for each diastereomer and sampler configuration, given that $k_A = R/A_{PUF}$. For the part-sheltered configuration these were 0.044, 0.049, and 0.050 cm s^{-1} for α-, β-, and γ-HBCD, respectively, and for the fully sheltered configuration – 0.028, 0.029, and 0.029 cm s^{-1} for α-, β-, and γ-HBCD, respectively. These can be used to estimate passive air sampling rates for the same sampler configuration but fitted with PUF disks of different macro surface areas.

Finally, the passive sampler derived concentrations are approximately one third (Table 8.1.1) lower than those derived using high-volume active air samplers (sum of both vapour and particulate phases).

Conclusions

- The majority (~65%) of each HBCD diastereomer is present in the vapour phase of indoor air.
- PUF disk passive air sampling devices may be deployed to monitor concentrations of HBCDs in indoor air.
- The concentrations reported in the three offices monitored in this study exceed substantially those reported elsewhere for outdoor air in the USA (Hoh & Hites, 2005), but are in line with those reported from Sweden (Remberger *et al*., 2004).

Discussion

The concentrations reported here (Table 8.1.1) exceed substantially those reported in outdoor air from the USA (Hoh & Hites, 2005) in 2002–03 (range 2.1–11 pg ΣHBCD m^{-3}). This apparent indoor:outdoor gradient may be attributable to the greater European usage of HBCD – two samples of

outdoor air from Stockholm sampled in 2000–01 contained 76 and 610 pg ΣHBCD m^{-3} (Remberger *et al*., 2004) – but may indicate that a substantial indoor:outdoor gradient exists for HBCDs, similar to those observed for related contaminants with extensive indoor use patterns like PCBs and PBDEs (Harrad *et al.*, 2006).

The HBCD isomer distribution pattern reported here (~65% γ-, 20% α-) more closely reflects that observed in the technical HBCD formulations; those in the US outdoor samples displayed a far greater abundance of α-HBCD (32–81%) in five of the seven samples reported.

The predominantly γ-HBCD pattern observed in indoor air differs from that observed in our study of indoor dust samples (Abdallah *et al.*, 2008), where 14–67% (average 32%) of ΣHBCD was the α-diastereomer.

There is no obvious explanation for the disparity between our results regarding the vapour:particle phase distribution of HBCDs and that of US outdoor air (Hoh & Hites, 2005), as the US outdoor samples included some taken in summer, thus eliminating the possibility that the absence of vapour phase HBCDs in the US samples was temperature-related.

The differences between HBCDs concentrations derived from passive and high-volume active samplers (Table 8.1.1) may be due partly to the difference in the monitoring periods. However, it is evident that the PUF disk-derived concentrations approximate very closely to the concentrations recorded in the vapour phase only by the high volume sampler. This indicates that the PUF disk samplers 'capture' only those HBCDs associated with the vapour phase, and suggests that PUF disk samplers may not be appropriate for use at low temperatures where the majority of airborne HBCDs may be expected to reside in the particulate phase.

Acknowledgement

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8.2 Tetrabromobisphenol-A (TBBP-A) in air and dust from Birmingham, UK: Implications for human exposure

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Background and objectives

TBBP-A is one of the most widely used brominated flame retardants (BFR) with a total worldwide production volume of 170 000 tonnes in 2004. It is used mainly as a reactive flame retardant covalently bonded to the polymer matrix in epoxy and polycarbonate resins used in printed circuit boards and electronic equipment. It can also be used as an additive, for instance in high impact polystyrene (HIPS) and acrylonitrile–butadiene–styrene (ABS) resins. The additive usage of TBBP-A is estimated to account for about 10% of its total applications. Due to its low solubility in water and low vapour pressure of 6.24×10^{-6} Pa, TBBP-A is likely to be associated with suspended particulate matter once released into the environment (Munn *et al*., 2006). It has been detected in air samples from Sweden (Sjodin *et al*., 2001) and Japan (Inoue *et al*., 2006). However, there are no reports of TBBP-A concentrations in air from the UK. TBBP-A has been quantified in indoor dust in a small number of samples from UK houses and offices in the European Parliament building (Santillo *et al*., 2003), but as yet there has been no assessment of human exposure arising from ingestion of such dust. This is a potentially important omission, given evidence of the importance of indoor dust ingestion as a pathway of exposure to other BFRs (Abdallah *et al*., 2008; Harrad *et al*., 2008).

The objectives of this study are:

- to report – for the first time – on the concentrations of TBBP-A in outdoor air and indoor air from different microenvironment categories in Birmingham, UK
- to report on TBBP-A concentrations in indoor dust from different microenvironment categories, including the first report on contamination in vehicular microenvironments
- to estimate the daily exposure to TBBP-A of UK adults and toddlers via air inhalation and dust ingestion.

Study description

Air samples were collected between June and December 2007 from homes (living rooms, n = 5), offices (n = 5) and public microenvironments (PME; three pubs and one restaurant) within the West Midlands conurbation. Outdoor air sampling was performed at the Elms Road Observatory Site (EROS) on the University of Birmingham campus.

Low volume active air samplers were used for air sampling. A double inlet pump (Capex L20X) was operated for 24 hours at a flow rate of 39 L min^{-1} to yield a sample comprising approximately 56 m^3 air. The particulate phase was collected by employing a 47 mm membrane filter (1.0 μm pore size, Whatman) housed in a standard open-face 47 mm filter holder. The designated flow rate was adjusted and maintained using a flow meter (Platon 50 L min^{-1}) connected to an adjustable valve. The flow meter was calibrated using a Gilibrator airflow calibrator (Gilian) which is classified as a primary standard device.

Dust samples were collected between September 2006 and June 2007 using a Nilfisk Sprint Plus 1600 W vacuum cleaner according to a clearly defined standard protocol (Harrad *et al*., 2008) from homes (living rooms, n = 34), offices (n = 28), PME (three pubs and one restaurant) and cars (n = 20) within

the West Midlands conurbation and in Basingstoke, Hampshire, in the south of England. Prior to analysis, dust samples were sieved through a 500 μm mesh size sieve.

Air samples were Soxhlet extracted while dust samples were extracted using pressurised liquid extraction (Dionex, ASE 300). The extracts were purified by washing with H_2SO_4 and Florisil chromatography, prior to analysis using LC/ESI/MS/MS.

Results

Table 8.2.1 summarises the concentrations of TBBP-A in the air samples studied. Despite the extensive usage of TBBP-A, its concentrations in indoor air are slightly lower than those of additive brominated flame retardants such as polybrominated diphenyl ethers (PBDE; Harrad *et al*., 2006).

Table 8.2.1 Summary of TBBP-A concentrations (pg m^{-3}) in air from the studied microenvironments

	Average	Standard deviation	Median	Minimum	Maximum
Homes (n = 5)	16	5	15	9	22
Offices (n = 5)	16	12	11	4	33
PMEs (n = 4)	26	7	27	17	32
Outdoor (n = 5)	0.76	0.06	0.74	0.69	0.85

The reported indoor air concentrations of TBBP-A in this study are in agreement with previous reports of TBBP-A concentrations in offices and lecture halls from Sweden (Sjodin *et al*., 2001) but lower than those reported from offices and houses from Japan (Inoue *et al*., 2006).

The concentrations of TBBP-A in indoor dust are summarised in Table 8.2.2. Concentrations in office dust reported in this study are a little higher than those detected by Santillo *et al*., 2003, in office dust from the European Parliament building, where concentrations in nine of 16 samples where TBBP-A was detectable were between 5 and 47 ng g^{-1}. Those in domestic dust are in agreement with those reported by Santillo *et al*. who reported TBBP-A levels ranging from 190 to 340 ng g^{-1} in four of 10 pooled samples of UK domestic dust.

Table 8.2.2 Summary of TBBP-A concentrations (ng g^{-1}) in dust from the studied microenvironments

	Average	Standard deviation	Median	Minimum	Maximum
Homes (n = 35, non-detects = 1)	87	71	62	<MDL[a]	380
Offices (n = 28, non-detects = 4)	49	46	36	<MDL[a]	140
PMEs (n = 4)	220	140	230	52	350
Cars (n = 20, non-detects = 10)	6	8	2	<MDL[a]	25

[a] Method detection limit (MDL) = 0.05 ng g^{-1}

Overall, the concentrations of TBBP-A found in indoor dust samples in this study demonstrate that despite its substantial production and use, TBBP-A is present at much lower levels than those reported elsewhere for PBDEs (Harrad *et al*., 2008) and hexabromocyclododecanes (HBCD; Abdallah *et al*., 2008). The absolute levels and relative magnitude of human exposure to TBBP-A via the ingestion of indoor dust and inhalation of indoor air are summarised in Table 8.2.3, alongside the recent Food

Standards Agency estimate of dietary exposure (<112 ng day^{-1} for a 70 kg adult, and <70 ng day^{-1} for a 10 kg toddler).

Table 8.2.3 Summary of estimates of exposure (ng day^{-1}) of UK adults and toddlers to TBBP-A via air, dust and diet, and relative significance (%) of each pathway

Intake (ng day^{-1})	Adult		Toddler (6–24 months)	
	air	dust	air	dust
5th %ile	0.16	0.42	0.03	1.07
Average	0.30	1.48	0.06	4.31
Median	0.27	1.17	0.05	3.28
95th %ile	0.44	3.24	0.09	8.52

% contribution						
	air	dust	diet[a]	air	dust	diet[a]
5th %ile	0.22	0.37	99.41	0.03	1.73	98.24
Average	0.31	1.42	98.27	0.13	5.84	94.03
Median	0.29	1.13	98.58	0.11	4.48	95.41
95th %ile	0.33	2.76	96.91	0.12	10.78	89.10

[a] UK Food Standards Agency (2006)

Conclusions

- TBBP-A was found to be present primarily in the particle phase rather than in the vapour phase of the air samples studied. Hence, passive air sampling devices that sample primarily the vapour phase only (e.g. PUF disk samplers) are not appropriate for monitoring TBBP-A in air.
- Concentrations of TBBP-A in indoor air exceed by an order of magnitude those in outdoor air.
- Because the majority of TBBP-A is covalently bonded within treated products, we hypothesise that it is far less susceptible to environmental release than its additive flame retardant counterparts, PBDEs and HBCDs.
- Although dietary exposure may currently be overestimated (no samples analysed by the Food Standards Agency contained TBBP-A above the detection limit), diet appears currently to be the principal human exposure pathway to TBBP-A (Table 8.2.3), followed by the ingestion of indoor dust.

Discussion

Higher concentrations of TBBP-A in indoor compared with outdoor air are consistent with the presence of substantial indoor sources. The low vapour pressure of TBBP-A is likely to be the cause of its preferential partitioning to the particulate rather than the vapour phase of air and for the relatively greater contribution to human exposure of indoor dust compared to indoor air. Diet represents the major human exposure pathway for TBBP-A (Table 8.2.3). This contrasts with the outcome of similar exposure assessments conducted for additive flame retardants like PBDEs and HBCDs where ingestion of indoor dust is the predominant exposure pathway for a substantial proportion of the population. As outlined above, we attribute this to the fact that TBBP-A is incorporated within treated products as a reactive flame retardant. Where consistent with achieving adequate flame retardancy, this may point to environmental benefits for the use of TBBP-A and similar 'reactive' flame retardants rather than their additive counterparts.

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8.3 Tools for smoke deposition measurement in the human lung

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Smoke from combustion is an important contributor to particulate air pollution and comprises a complex and dynamic matrix of gaseous compounds and particulate material. Smoke particles are typically small, in the region of 30–150 nm count median diameter (CMD) for high-efficiency combustion, for example automotive emissions, or 100–250 nm for less-efficient combustion such as biomass burning or tobacco smoke. The deposition efficiency of particles of these sizes has proven difficult to measure, particularly where high concentration and the hygroscopic nature of the smoke droplets mean that this particle diameter may change rapidly via coagulation and evaporation or condensation. The chemical composition and partition between vapour and particle phases of smoke is equally complex, can change continuously and is strongly influenced by time, temperature, chemistry and dilution of smoke.

The magnitude and site of deposition of inert particles at low concentrations is well known and has been modelled by the International Commission for Radiological Protection (ICRP, 1994). However, greater relative deposition efficiencies of 60–80% in the lung have been reported for mainstream tobacco smoke (Baker & Dixon, 2006) and approximately 40% for environmental tobacco smoke (Strong *et al.,* 1994; McAughey *et al.,* 1995; Morawska *et al.,* 2005).

Recent advances in measurement allow these dynamic properties of smokes to be better quantified. The use of fast electrical mobility spectrometers has allowed particle size measurement in the range 10–1000 nm at 10 Hz resolution (Reavell *et al.*, 2002) compared with previous electrical mobility techniques, which required 60–90 second scan times. We also observe an improvement in comparison to light scattering based measurement systems, which generally do not measure below 300 nm CMD. Measurements at our laboratory have shown inhaled particle size for mainstream tobacco smoke to be in the range of 130–260 nm CMD. Exhaled smoke has been measured in the range of 200–300 nm CMD, supporting the hypothesis that deposition is driven by Brownian motion, with large increases in retention from greater depth of inhalation relative to tidal breathing, and increasing breath-hold duration between inhalation and exhalation. Puffing and inhalation behaviour have been measured separately with non-invasive techniques.

Chemical measurement of the particle and vapour phases has been addressed by utilising time-of-flight mass spectrometry, combined with two selective photo-ionisation techniques, avoiding complication of the spectra by mass fragments. The resonance-enhanced multi-photon ionisation (REMPI) technique uses at least two ultraviolet (UV) photons for photo-ionisation, which takes place via an optical resonance absorption step. In practice, REMPI is highly sensitive for aromatic compounds. The single photon ionisation (SPI) technique uses vacuum ultraviolet (VUV) photons for ionisation. With SPI additional compounds, for example aliphatic hydrocarbons, carbonyl compounds and nitrogen-containing substances, are accessible. Measurement rates of up to 20 Hz are possible, although in practice a 3-Hz rate has been used for improved counting statistics.

Initial mass spectrometry data show that most mainstream smoke constituents feature a continuous increase from the first to the last puff. There are some substances, in particular unsaturated hydrocarbons – for example butadiene, isoprene, and propyne – which have proportionately higher amounts in the first puff (Mitschke *et al.*, 2005). However, principal component analysis shows that the chemical mixture of the particle is relatively insensitive to changes in particle diameter.

In conclusion, the use of time-resolved analytical techniques is offering fresh insight into the chemical and aerosol behaviour of tobacco smoke, thus improving our understanding of regional deposition and

dose in the lung. Characterisation of these dosimetric processes offers scope for better understanding of sites and causes of disease, with appropriate dose application to *in vitro* models of disease. The techniques described are equally suitable for measuring regional deposition and dose of environmental particle exposure from all combustion sources.

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8.4 Correlation between microenvironment and personal exposure levels of nitrogen dioxide: Research and policy implications

(Marguerite) Jane Newbold[1], Ranjeet Sokhi[1], Chiraporn Kornartit[1], Agneta Burton[2] & Ravindra Khaiwal[1]

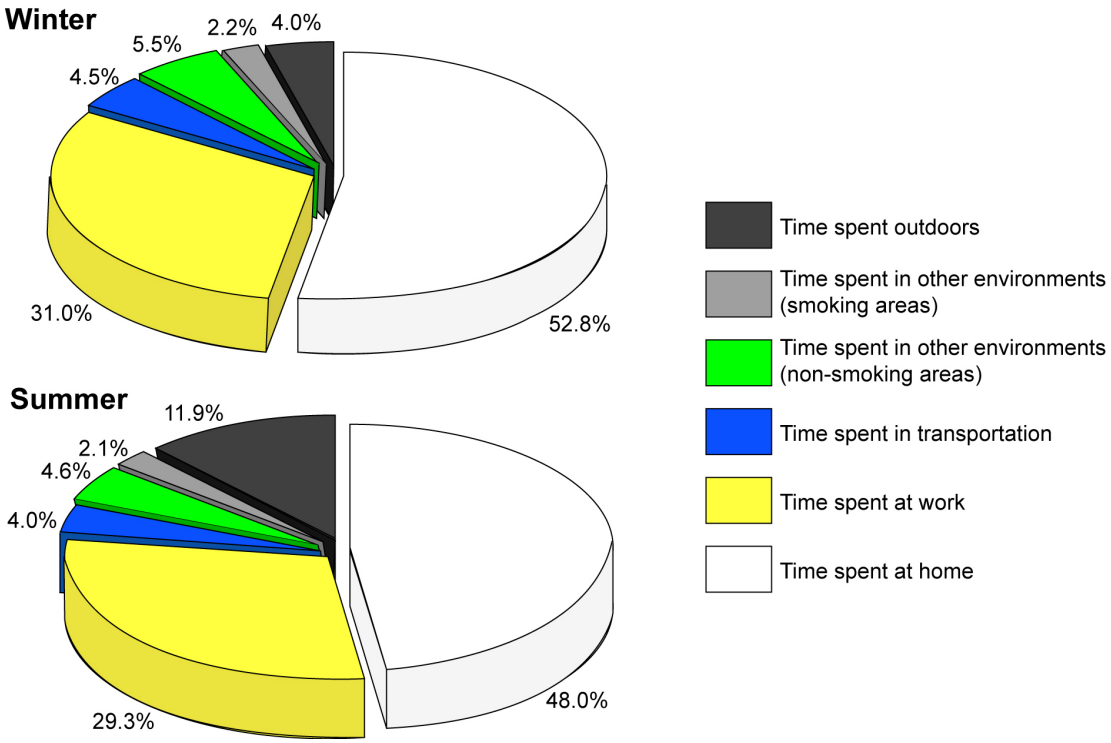
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Background and objectives

In 2003 the European Commission (EC) adopted a new strategy on environment and health with the overall aim of reducing diseases caused by environmental factors. The EU Action Plan on Environment and Health (European Commission, 2004) is an initiative that recognises the importance of integrated strategy on air pollution which considers not only outdoor air quality, which is quite well regulated, but also includes possible solutions concerning indoor pollutants. Guidelines have been established for outdoor air quality but so far no guidelines have been developed for indoor air quality, despite the fact that most people spend over 80% of their time indoors (Figure 8.4.1).

More than 900 organic compounds have been detected in indoor air (SCALE, 2004), plus particulates and biological materials, all of which may have irritating and sensitising potential.

Figure 8.4.1 Time spent in micro environments during a 7-day period of exposure (Kornartit, 2005)



The EC Scientific Committee on Health and Environmental Risks (SCHER) was asked to provide a basis for assessment of risks to human health from indoor air quality and a sound scientific basis for the development and implementation of policies. Taking into account the outcome of the INDEX

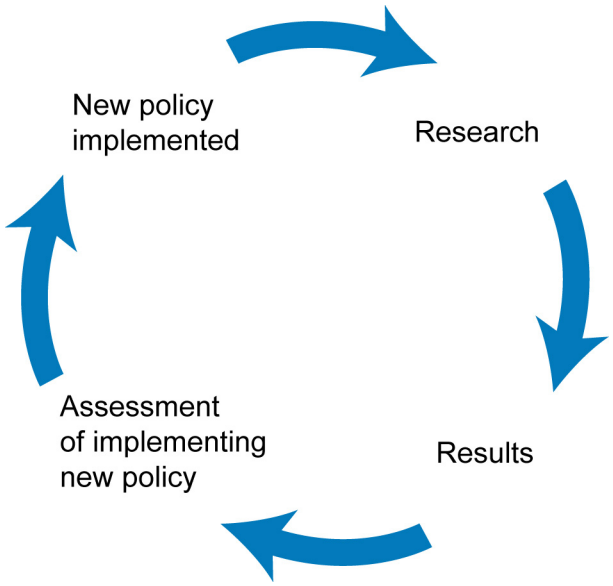
report by DG JRC (Kotzias *et al.,* 2005) a list of compounds of the highest concern based on health impact criteria has been produced. The highest priority chemicals were determined to be formaldehyde, carbon monoxide, nitrogen dioxide, benzene and naphthalene (SCHER, 2007).

Studies such as this on sources and health effects of exposure to pollutants are needed before new policies can be adopted to protect the community.

Indoor air quality and nitrogen dioxide

Over 50% of total NO_2 comes from traffic (DoE, 1995), but a study in the UK suggests 70–75% of personal exposure to NO_2 occurs within the home (Raw & Coward, 1992). It is important to establish the source of this indoor NO_2, the seasonal occurrence and the influence of outside NO_2 concentrations, as all have to be taken into consideration when determining whether new policies can be brought in to protect the public from any health consequences. This process of research leading to new policy initiatives is a dynamic one with assessment and review of new policies, including cost–benefit analysis, leading to further research and amended policies (Figure 8.4.2).

Figure 8.4.2 Continual assessment and research supporting new policy initiatives



Study description

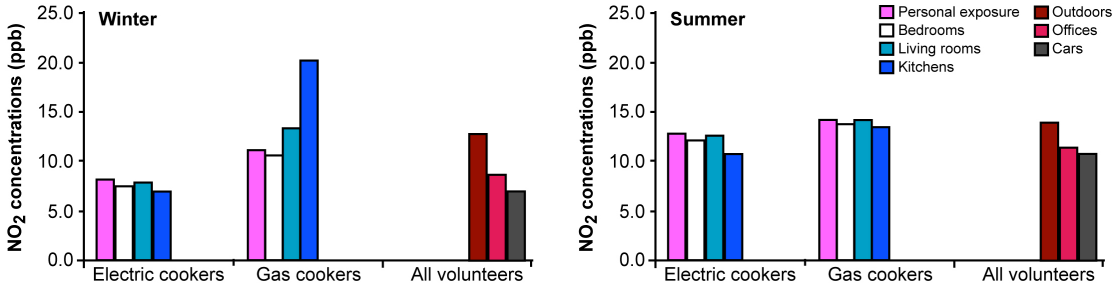
A study was carried out in Hertfordshire in the winter of 2000 and the summer of 2001 which targeted a group of 60 randomly selected office workers living and working in an urban area and with ages ranging from 21 to 60 years.

The 60 office workers were asked to fill in activity diaries and questionnaires. At the same time, weekly average NO_2 concentrations for personal exposure and fixed indoor and outdoor micro-environments were measured using Palmes passive NO_2 diffusion tubes (Palmes *et al.,* 1976). Seven locations were selected for these measurements: personal, bedroom, living room, kitchen, front door (outside house), office and inside car, and two passive Palmes diffusion tubes were used at each site. During the same periods, correlations between the weekly personal exposures and mean indoor and outdoor concentrations were made. All tubes were prepared an analysed at the University of Hertfordshire.

Results

The weekly average NO_2 concentrations in all microenvironments are shown in Figure 8.4.3. In the winter campaign it is shown that gas appliances are a main factor affecting the level of indoor NO_2 with all rooms showing elevated NO_2 levels. Kitchens with gas cookers had NO_2 levels two to three times higher than were found in kitchens with electric cookers. The summer campaign shows NO_2 levels generally higher than those recorded in the winter campaign indicating the influence of outdoor air quality. For all volunteers, the average personal outdoor exposure to NO_2 concentrations in both the winter and summer campaigns is similar.

Figure 8.4.3 Weekly average NO_2 concentrations in all microenvironments for volunteers using electric and gas cookers



Source: (Kornartit, 2005)

Other results not detailed here show the time weighted average exposure plotted against personal exposure to NO_2 concentrations for the winter and summer campaigns. In the winter campaign, good correlation was found between personal exposure and microenvironment, while in the summer campaign the correlation was not so strong (Kornartit, 2005).This difference could be due to more time being spent outside in the summer, more windows or doors being open and increasing ventilation, and volunteers being involved in different activities.

Discussions and conclusions

The results from this research have been used to highlight the need for more work on exposure, especially indoor exposure, and the need to set some indoor air quality guidelines, possibly linked to type of premises (e.g. school, college, hospital, office) and the season of the year.

Ventilation plays a part in influencing indoor air quality. In winter windows are generally kept closed and people stay indoors for longer; however in summer, with more ventilation, the influence of outdoor air quality is much greater. In urban areas, proximity to traffic and wind direction play a key role. Naturally ventilated buildings offer virtually no protection from infiltration from outside air but ventilation design taking into account the surrounding buildings and street canyons may help future developers.

Results from the NO_2 research described here for the winter and summer campaigns show the importance of personal exposure and microenvironment in determining levels of a pollutant which may cause adverse health effects. If new policies and regulations are to be drawn up to control indoor air quality, more research of this type on personal exposure needs to be done.

These results re-enforce the findings that stricter regulations may be needed for flue-less gas appliances to ensure NO_2 levels are kept at safer levels in any indoor environment.

Personal exposure estimates are necessary for health studies and policy development, with more personal exposure research linked to evidence-based health risk assessment. This type of work has special significance to those people who spend most of their time indoors, for example the elderly.

Improved assessment methods may be necessary to determine the effectiveness of air quality measures on personal exposure levels.

Further work on linking policy implications to research needs related to air quality and health is being carried out in the CAIR4HEALTH project (an EU funded Framework 6 project). Research needs and gaps in knowledge related to air quality and health were identified from interviews with experts and from detailed review of key publications, such as position papers, strategy documents, workshop reports and reviews of needs. The first report is now with the Commission (Newbold *et al.,* 2008).

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8.5 Intercomparison of particulate matter monitoring devices

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Background and objectives

Due to the health effects and climatic influence of airborne particulate matter (PM) pollution, there is a requirement by legislation to monitor the levels of the PM_{10} fraction in all relevant exposure locations for comparison against air quality standards. The measurements should be carried out using the reference method, as defined in the European Committee on Standardisation standard EN12341, or equivalent. PM_{10} monitoring in the UK is mainly based on the Tapered Element Oscillating Microbalance (TEOM) analyser, although it has recently been shown that this is not equivalent to the reference method (Harrison *et al.*, 2006).

The measurement devices that are equivalent to the reference method (e.g. the Partisol 2025 Sequential Sampler and TEOM retrofitted with a filter dynamics measurement system) tend to be expensive and immobile. Cheaper and more portable instruments are available, such as the Optical Scattering Instantaneous Respirable Dust Indication System (OSIRIS) monitor, and these could be used to provide preliminary wide-area assessment of airborne PM. Further benefits of the OSIRIS monitor are its high time resolution and ability to simultaneously measure data for PM_{10}, $PM_{2.5}$ and PM_1 fractions. Being able to measure the fine PM fraction is relevant to the increased interest in its behaviour and effects. A drawback of the OSIRIS monitor is the fact that its response is not directly related to mass concentration and the associated calibration depends on assumptions of particle distribution and composition, which may vary. It is therefore necessary to compare the OSIRIS monitors with mass-responsive devices to determine the validity of these assumptions.

The purpose of this study was to undertake a co-located instrument intercomparison of OSIRIS vs TEOM (PM_{10}) and OSIRIS vs OSIRIS (PM_{10}, $PM_{2.5}$ and PM_1) monitors, to determine their level of agreement and therefore assess the suitability of the OSIRIS monitor for measuring PM in the UK.

Study description

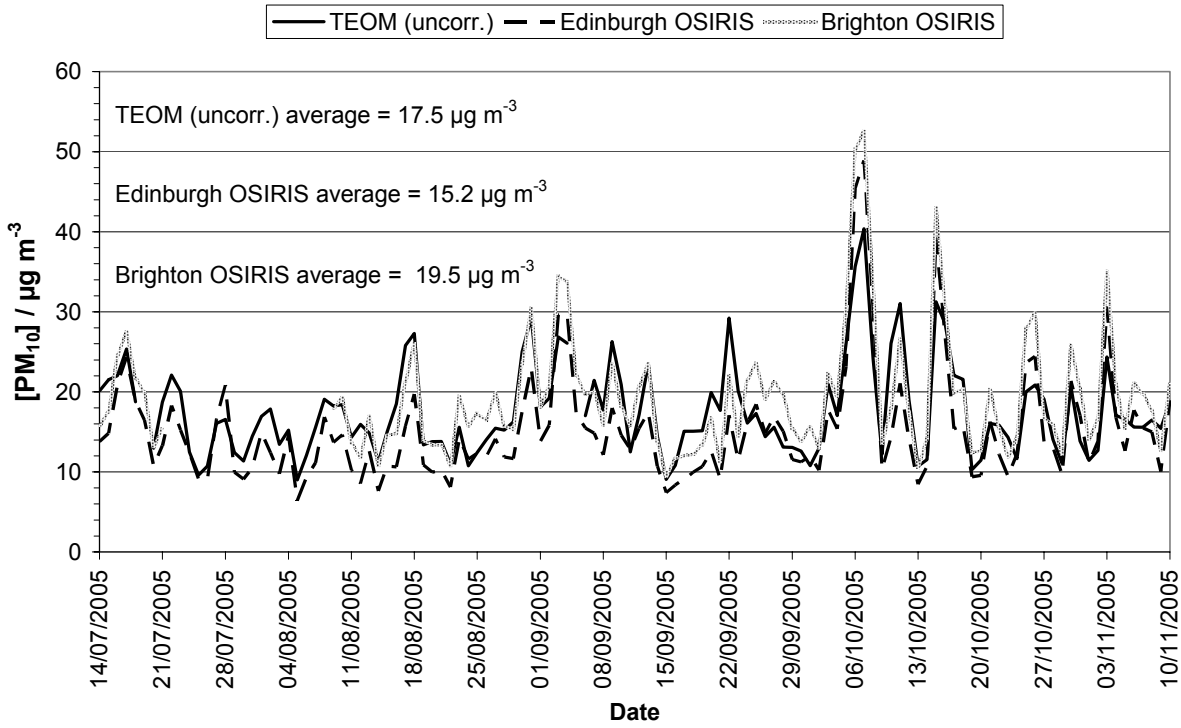
The study was carried out from 14 July to 10 November 2005, with three monitors co-located at the Automatic Urban and Rural Network site in Horley (urban background; OS grid reference: 528203, 142431), approximately 1.5 km from the runway of Gatwick airport. The following instruments were compared: a TEOM monitor fitted with a size-selective PM_{10} head, an OSIRIS monitor from the University of Edinburgh ('Edinburgh OSIRIS') and an OSIRIS monitor on loan from Brighton ('Brighton OSIRIS').

Semi-continuous data of PM concentration ($\mu g\ m^{-3}$) were collected from these instruments for comparison. The hourly TEOM PM_{10} measurements were averaged to daily values (midnight to midnight) provided there was at least 90% data capture (22 1-hour measurements). The 15-minute measurements of PM_{10}, $PM_{2.5}$ and PM_1 from the OSIRIS monitors were averaged to daily values, again provided there was at least 90% data capture.

Results and discussion

Time-series data of PM_{10} concentration from the three monitors are shown in Figure 8.5.1. The TEOM values are reported as 'TEOM (uncorr.)' since the 1.3 correction factor used in the UK monitoring network has not been applied.

Figure 8.5.1 Time series of daily mean PM_{10} measurements; average values are the mean concentrations over the whole study period



It can be seen that the general trends in daily PM_{10} concentration were followed by each of the instruments throughout the period of measurement, although the mean concentrations of the OSIRIS monitors over the whole study period differed from those of the TEOM by up to 13%.

Comparisons between the daily mean PM_{10} concentrations of the two OSIRIS monitors (Figure 8.5.2) show they had a very good linear correlation as indicated by the R^2 value of 0.98. The line from the major axis estimation indicates that, although there was very good agreement between the two monitors, the Brighton OSIRIS consistently over-read the Edinburgh OSIRIS. As this systematic bias is linear it could easily be corrected.

Comparison of both OSIRIS monitors against the TEOM (Figure 8.5.3) shows reasonable linear correlation, with R^2 values of 0.7. The major axis lines both have slopes >1 and intercepts <1. This indicates a tendency for the OSIRIS monitors to under-read the TEOM at low PM_{10} concentrations, and over-read it at higher concentrations. Again, this systematic bias is linear so it could be corrected, subject to the uncertainty demonstrated by the scatter in the relationships.

The linear correlation between the two OSIRIS monitors for $PM_{2.5}$ and PM_1 was almost perfect, as indicated by the R^2 values of 1.00, and the slopes and intercepts of the major axis lines being close to 1 and 0 respectively (Figure 8.5.4). However, a correction factor could be applied to the PM_1 data to account for the slight systematic under-reading of the Edinburgh OSIRIS.

The mean hourly ratios of $PM_{2.5}$:PM_{10} and PM_1:PM_{10}, as measured by the OSIRIS monitors, were ~0.45 and ~0.13 respectively. Assuming the OSIRIS calibration is reliable, this suggests, in general terms, that ~45% of the PM in PM_{10} during this study was predominantly of direct anthropogenic origin and ~55% was predominantly of natural or indirect anthropogenic origin (Heal *et al.*, 2005).

Figure 8.5.2 Comparison of daily mean PM_{10} concentrations between the two OSIRIS monitors

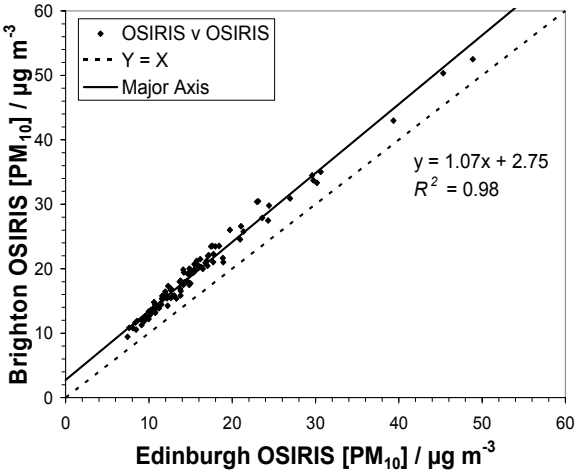
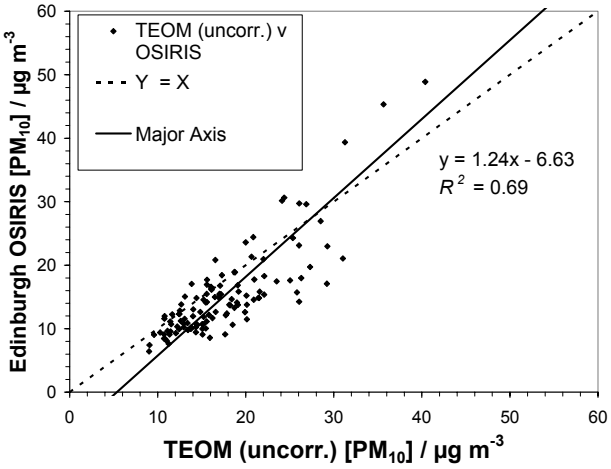


Figure 8.5.3 Comparison of daily mean PM_{10} concentrations between the TEOM and each OSIRIS monitor



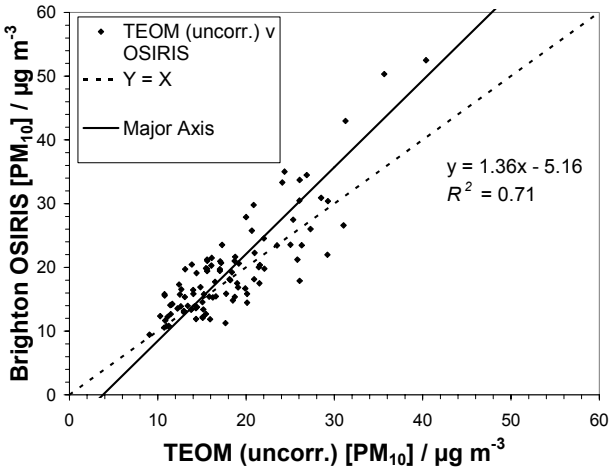
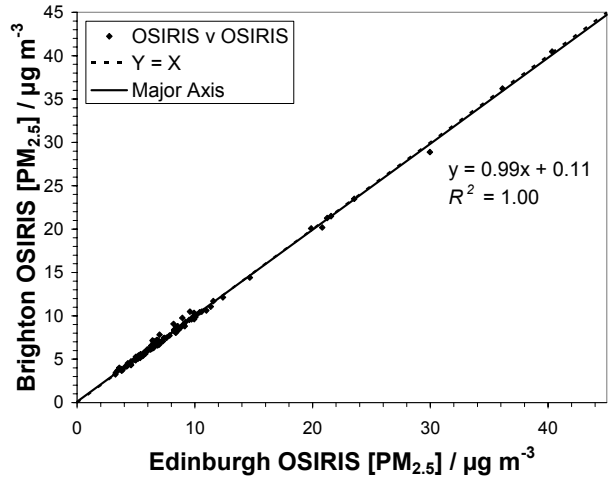
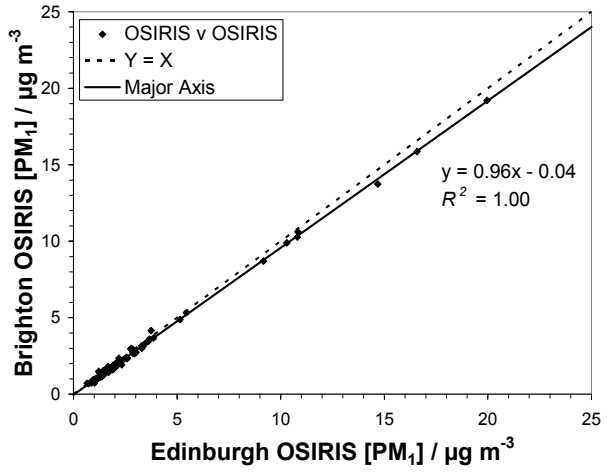


Figure 8.5.4 Comparison of daily mean $PM_{2.5}$ and PM_1 concentrations for the Edinburgh and Brighton OSIRIS monitors





Conclusions

- The trends in daily PM_{10} concentration as measured by the TEOM monitor were consistently matched by both OSIRIS monitors over the whole study period.
- There was good linear correlation of PM_{10} concentration for the OSIRIS versus OSIRIS and TEOM versus OSIRIS data. There was some systematic bias in these results but, since it was linear, it could easily be compensated for by using a correction factor.
- The two OSIRIS monitors were almost in complete agreement for $PM_{2.5}$ and PM_1 concentration values, showing a high level of precision for the measurement of these two metrics.
- The OSIRIS monitor shows good potential as a method for measuring PM concentration in the UK. However, further work will need to be done to compare it against the reference method, or equivalent, for PM_{10}, $PM_{2.5}$ and PM_1 to analyse this potential more rigorously.

Acknowledgements

The authors would like to thank Dr Iain Beverland and the Sussex Air Quality Steering Group for the loan of the OSIRIS monitors.

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8.6 Breach of the NO_2 standard due to roadwork-caused traffic congestion and associated abuse of the protocol on NO_2 diffusion tubes

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Friends of the Earth Cymru

Background and objectives

A severe failure of the short-term NO_2 standard recorded by the Vale of Glamorgan in 2006 was associated with year-long roadworks, which caused extended periods of congested traffic past the roadside monitoring location and nearby dwellings. Data from a continuous monitor and diffusion tubes, both nearby and co-located, are available. The former showed an annual average of 68 $\mu g/m^3$ but the authorities reported the figures from diffusion tubes after bias correction as up to 39 $\mu g/m^3$. We consider the implications for the diffusion tube protocol as well as for the general validation process and the need to take full advantage of the continuous chemiluminescent monitoring.

Traffic congestion NO_2 enhanced by roadworks

High NO_2 readings had been found from diffusion tubes at busy crossroads (Cogan roundabout) on a major access into Cardiff, south of the city and west of Cardiff Bay, but in an exposed location. Levels were above the limit of 40 $\mu g/m^3$ for annual NO_2 (Table 8.6.1). From 2002, diffusion tube monitoring on the road arm 350 m away into Penarth showed higher levels at times, despite lower traffic at ~24 000 vpd. This is critical, being near housing at the start of a canyon street which suffers peak-time traffic queuing (Penarth Times, 2006). In May 2005 a continuous monitor was installed on this road arm. Then major roadworks began in January 2006 for twelve months, to replace the roundabout with a crossroads junction with slip roads and light controls. The roadworks intensified the congestion and queuing, with consequences seen in NO_2 levels.

Table 8.6.1 Annual averages from monthly NO_2 diffusion tubes in the locality

Location[a]\Year	98	99	00	01	02	03	04	05	06	07
Cogan roundabout	44	44	41	40	38	39	42	36	32	37
Andrew Rd, Llandough						39	38	33	35	40
Corner, Windsor/Andrew Rd[b]								26	28	29
road-corner lamp post								29	31	26
Windsor Rd, Penarth					43	42	35	31	36	38
Windsor Rd, Penarth /Facade						38	34	31	34	35
160 Windsor Rd, Penarth							38	35	39	46
Bias correction factor					0.95	0.87	0.84	0.75	0.78	1.0

Source: Vale of Glamorgan Council (2007)

[a] Grid references for the sites are given in the report, Cogan roundabout is at E,N: 317431,172159.

[b] The continuous monitor and co-located diffusion tubes at the Corner Windsor/Andrew Rd (317550, 172482 ST175724) are given as 5.3 metres (actual 7–8 m) from the kerb (Vale of Glamorgan Council, 2007, Appendix 3).

That the NO_2 limit of 40 μg/m^3 would be exceeded could be predicted from the previous years' results[1]. Apart from 40 in March, monthly averages from the continuous monitor were 50–70 μg/m^3. By late summer it was also clear that the 1-hour NO_2 standard would be breached, as the number of hours exceeding 200 μg/m^3 mounted over the limit of 18 per year. However, the only decision by agreement with Welsh Assembly officials was to keep monitoring (not to re-schedule work to give up carriageway space for peak-hour traffic). The end-year data gave the annual average NO_2 as 68 μg/m^3, and 144 exceedances of the 1-hour standard of 200 μg/m^3 on 49 days, compared with the maximum 18 exceedances allowed in a full year (Vale of Glamorgan Council, 2007). Not until reports to committees in July 2007 and questions asked by Friends of the Earth did information emerge. The Council report quoted figures from diffusion tube monitoring, from which the levels given were as just under 40 μg/m^3 and not significantly worse than earlier years (Vale of Glamorgan Council, 2007). The issues to be addressed here are how diffusion tube data can be preferred over the continuous chemiluminescence results – the EU-standard – how reporting to the public failed, and whether any lessons have been drawn.

Bias correction for the NO_2 diffusion tubes

Errors in diffusion tube monitors can arise, for example from local positioning, because of traffic or wind-induced turbulence, blocking of UV radiation (which fixes the NO_2), or from interference of peroxyacetyl nitrate (PAN) produced under high ozone (AEA, 2006). Errors in preparation and deployment are checked by exposing tubes in triplicate, which gives a measure of the precision, though not of errors from handling the tubes before and after exposure. The tubes used from Cardiff Scientific Services (50% TEA in acetone) have mainly good laboratory precision (three times out of four in 2005–6). The major issue lies in assessing the bias. The bias differs between laboratories for reasons unknown, even when tube preparation technique, tube materials, and analytical techniques are broadly the same (AEA, 2006). The protocol for calculating bias of NO_2 diffusion tubes requires co-location of triplicate tubes with a chemifluorescence continuous monitor and derivation of a bias correction factor from the annual averages. Bias factors derived from all co-location studies with each laboratory's tubes are to be used (Table 8.6.2), taking the continuous monitors as standard.

Other laboratories than Cardiff use the 50% TEA in acetone tubes, particularly Gradko, for which there are many more co-location studies. These show both more consistency and mostly small bias (1 ± 0.05). Cardiff's large spread in bias (and few data per year) implies the bias corrections are less reliable, although no uncertainty range is required. AEA's Guidance (2006) says there are theoretical reasons why diffusion tube performance varies between roadside and urban background locations, so "it makes sense to use a roadside site for the co-location study" if used for the bias correction of roadside diffusion tube data. Thus the 'O' co-location results should be excluded and the Penarth co-location results included.

Monthly figures for the bias are available from the triplicate diffusion tube and continuous monitor data; they show the bias variation is greater than the diffusion tube precision, so can in principle help elucidate factors causing the bias. There is some suggestion of tube non-linearity (high bias factor for high NO_2) but some other factors must play a significant role (e.g. wind, humidity or ozone levels).

NO_2 results in Penarth

Table 8.6.1 shows the Council's bias corrections (final row); these differ from those in Table 8.6.2 mainly because Monmouth's 'I' was entered late. The actual bias factor (0.99, average of the two 2003 results) would set all four diffusion tube points for 2003 above 40 μg/m^3 (which may be why the Council report chose not to use 0.99). The corner Windsor/Andrew Rd is the site of the continuous monitor and this more open location does show lower readings than the other Windsor Rd tubes which are adjacent to dwellings.

[1] While NO_2 levels had been falling as lower NO_x engines came in under Euro-emission standards, the prompt-NO_2 problem was also becoming clear – that the fraction of NO_x emitted as NO_2 was increasing. Particle oxidising traps apparently oxidise the NO_x, Euro III diesel cars emit 20–70% NO_2 and the average fraction is increasing well above the 5% assumed (AQEG, 2007). This had been reported in the previous AQ review (Vale of Glamorgan, 2006).

Table 8.6.2 Bias factors for Cardiff Scientific Services' NO_2 tubes using the UWE database in the *LAQM tools* section

Site type	Local authority	Length of study months	Diffusion tube Mean conc. (Dm) µg/m³	Automatic monitor Mean conc. (Cm) µg/m³	Bias (B)	Tube precision	Bias adjustment Cm/Dm	Year
UC	Cardiff CC	11	34	32	6.2%	G	0.94	2002
UC	Cardiff CC	11	40	35	13.5%	P	0.88	2003
UC	Cardiff CC	12	37	31	22.5%	P	0.82	2004
R	Monmouthshire DC	12	51	37	37.1%	G	0.73	2005
I	Monmouthshire DC	12	30	34	-10.5%	G	1.12	2003
O	Vale of Glamorgan	11	15	11	33.1%	P	0.75	2005
UB	Cardiff CC	12	36	30	18.6%	G	0.84	2006
O	Vale of Glamorgan	11	15	12	29.6%	G	0.77	2006
UB	Monmouthshire DC	12	29	25	12.7%	na	0.89	2006
R	Monmouthshire DC	12	54	34	56.0%	na	0.64	2006

Source: University of the West of England (2008) www.uwe.ac.uk/aqm/review/diffusiontube290208.xls

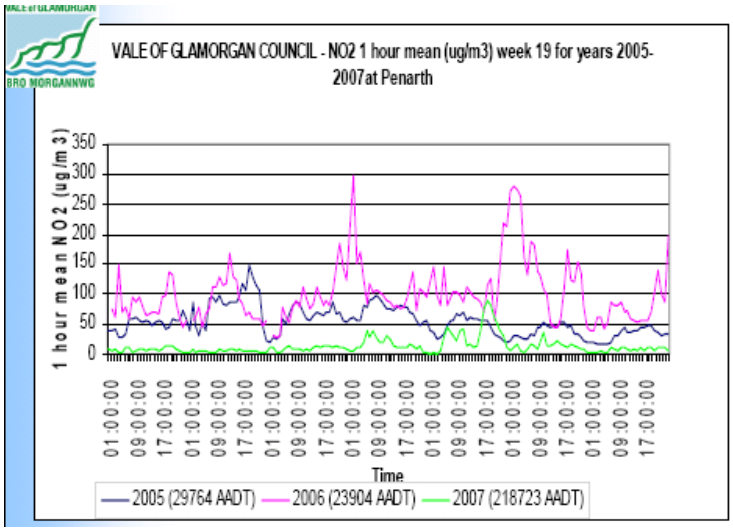
The bias factor varies substantially, both from year to year and between locations (UC, urban centre; UB, urban background; R, roadside; O, other; I, industrial). The first three bias factors from co-location with the AURN monitor in central Cardiff are quite consistent, but the two busy roadside studies by Monmouthshire found much lower bias factor (diffusion tubes over-reading). The two 'O' studies by the Vale of Glamorgan are very low exposures (as well as low bias factors). They are from a remote rural site under the airport takeoff path, which is also impacted occasionally by the Aberthaw power station plume (shown by SO_2 records). The Penarth co-location results (1.94 bias factor) are not entered on UWE's database.

Figure 8.6.1 gives continuous monitor data for the third week in May for the years 2005–7, showing characteristic diurnal variation with rises during morning and evening peaks, including two very strong rises in 2006 evening peaks, giving several hours >200 µg/m^3 (Choo Yin, 2007). Such peaks repeated through the summer, becoming worse during autumn mornings, when monthly averages exceeded 70 µg/m^3. However, nothing shows up in the diffusion tube averages of Table 8.6.1. This is doubtless due to faulty bias factors. If the protocol was properly followed, replacing the 'O' result for 2006 with the Penarth roadside result (1.94), then the average bias factor was 1.08 and the three Windsor Rd results increase from 34–39 to 47–54 µg/m^3. The bias factor for 2005 is uncertain, relying on the strangely low Monmouth 'R' because the Cardiff AURN co-location result was higher but covered only half the year.

Reporting and checking the data

The breach of the NO2 standard did not become public until data were presented to committee in mid-2007. The presentation (Choo Yin, 2007) stated compliance with the annual standard on the basis of the diffusion tube results (Table 8.6.1) and mentioned the 1-hour limit only as an 'objective'. In a number of emails, Friends of the Earth objected to the Council and the Welsh Assembly Government (WAG) Air Quality (AQ) unit and suggested that local residents had been exposed to unacceptable levels of pollution. However, after consultation with their consultants, WAG confirmed their approval of the report (Vale of Glamorgan 2007).

Figure 8.6.1 Data from the continuous monitor for the third week of May



Source: Choo Yin (2007)

Middle (heavy line), 2005; upper line, 2006; lower line, 2007 (windy period)

Checking the continuous monitor

Rather than investigate and reach an understanding of the exceptional bias factor (1.94) and diffusion tubes' under-reading, the anomaly has been dismissed as due to temporary traffic congestion. Yet the AEA Guidance (2006) says data quality is of 'paramount importance', so good QA/QC procedures must be applied to the automatic monitoring.

One issue is the siting of the standard monitor – at a side road junction and within metres of car parking that is used for a taxi business. It is 7–8m from the major road edge (stated as 5.3m from the kerb), so does not comply with the 1m to 5m specified, and is close and is close (4.4m) to cars stopping and emerging from the side road. It is sited at the corner of fencing where eddies occur during windy conditions, which diffusion tubes are supposed to avoid (AEA, 2006; European Council, 1999). The site is open and not representative of local sites where people are exposed – and to pollution levels known to be higher in the 'canyon' stretch of road. A look at the detailed data readily finds anomalies, for example some peaks overnight or unexpectedly early in the morning (cf. peaks at 1.00 am in Figure 8.6.1) and odd peaking out of phase with NO data. Interference from ozone or interior condensation has been suggested.

Conclusions

The continuous monitor provided specially for this high NO_2 location is badly sited, apparently in disregard of guidance and subject to interfering sources. While some runs of high readings by the monitor are suspect, no action appears to have been taken to query the misreadings and correct the record. Instead of proper use of its results as standard and for calibrating the roadside diffusion tubes, the diffusion tube data with questionable corrections were given preference. 'Bias corrections' from rural sites included in the *LAQM tools* database are anomalously low, unsuited for roadside diffusion tube corrections, but are still used for adjusting the NO_2 data. If the high result from Penarth's co-location study had been properly included, corrected data would have been significantly higher.

Despite the standard monitor's poor siting in a less polluted location, the indicated breach of the NO_2 standard due to roadworks-caused traffic congestion became clear midway through 2006. However no action appears to have been taken, nor were the affected residents informed. This episode spotlights a need to take into account traffic pollution when organising and scheduling roadworks, in order to ensure compliance with the statutory AQ standards.

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8.7 Constructing and validating modelled concentration surfaces for black smoke and sulphur dioxide across GB, 1955–2001

Chloe Morris[1], Anna Hansell[1], John Gulliver[2], Danielle Vienneau[1] & David Briggs[1]

[1]Imperial College, London, [2]University of the West of Scotland, Paisley

Background and objectives

The aim of this project is to generate a retrospective time series of annual average black smoke and sulphur dioxide (SO_2) concentrations for Great Britain, from 1955–2001, as a basis for exposure assessment that can be applied to existing British cohort studies, in particular as part of the Chronic Health Effects of Smoke and SO_2 in the UK (CHESS-UK) project. To do this, various GIS methodologies are being employed.

Study description

This abstract relates to methodology currently under development to estimate historical spatial distributions of black smoke and SO_2 concentrations for key years across the study period (1961, 1971, 1981, 1991, 2001). Monitored black smoke and SO_2 levels from the Smoke and Sulphur Dioxide National Survey, comprising 3100 monitoring stations in operation over the time period, are used as a set of core data. Ordinary kriging of monitored annual black smoke and SO_2 concentrations for sample years (1962, 1971, 1981 and 1991), including data from stations with concentrations available for ≥75% of days in the year, was conducted to provide a 'benchmark' method.

Results

Ordinary kriging models were developed using 75% of the data with remaining data reserved for validation. The statistics are shown for SO_2 and black smoke across sample years in Tables 8.7.1 and 8.7.2, respectively.

Table 8.7.1 Validation statistics for ordinary kriging models for SO_2 in sample years

Sulphur dioxide	1962	1971	1981	1991
No. training sites	453	693	711	143
No. validation sites	151	232	238	48
R^2	0.59	0.64	0.47	0.46
RMSE	49.87	27.94	15.67	11.35

Table 8.7.2 Validation statistics for ordinary kriging models for black smoke in sample years

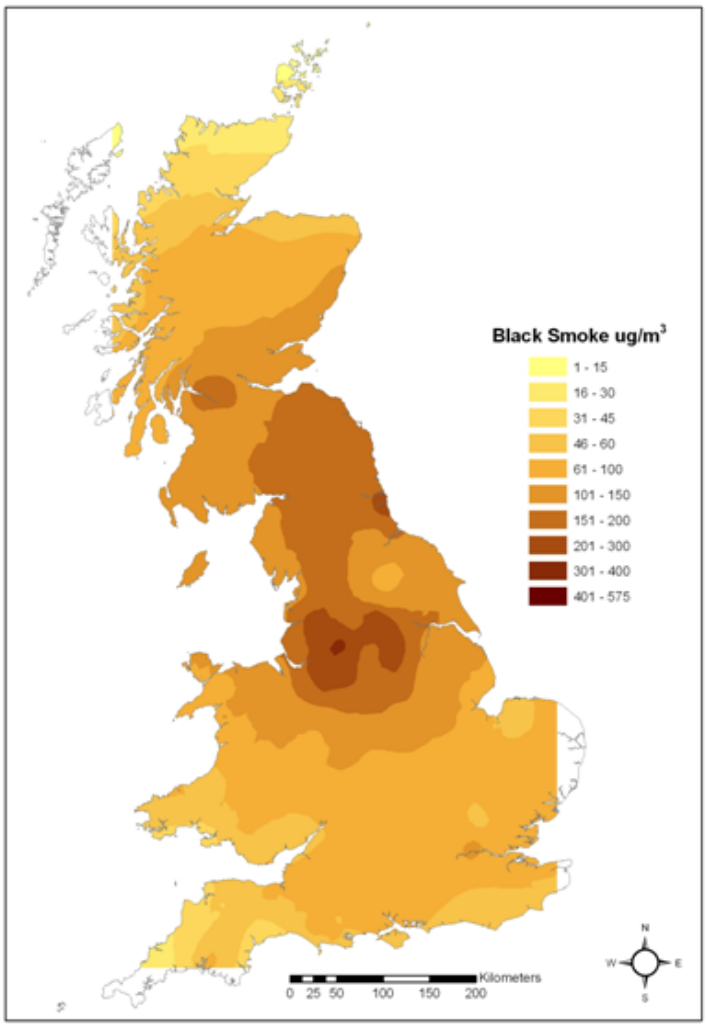
Black smoke	1962	1971	1981	1991
No. training sites	500	724	726	144
No. validation sites	167	242	242	48
R^2	0.64	0.66	0.50	0.38
RMSE	49.26	18.83	7.88	6.63

Models worked well for 1962 (Figure 8.7.1) and 1971 for SO_2 and black smoke (r^2 values ranging between 0.59 and 0.66) but were less reliable for later years (r^2 values between 0.38 and 0.50) when monitoring station numbers fell from around 700 to less than 200.

Highest concentrations were consistently observed in northern England (Liverpool, Manchester and Tyneside), with lowest values in Scotland and the southwest. Prediction errors were not evenly distributed and error maps showed that the greatest errors occurred where there was greater variation among sites and where there were few monitoring sites. Concentrations of both pollutants declined markedly over time, with 90th percentile values for black smoke falling from 270 μg/m^3 in 1962 to 25 μg/m^3 in 1991.

Additional GIS methods currently being evaluated include regression-based methods and affinity zone stratification. In initial trials these methods have shown to have good potential where appropriate covariate data is available. A range of input data has been investigated and a small-area database of geo-referenced data across the time series has been compiled as a basis for air pollution modelling. Data include emission maps (generated with AEA Technology Ltd for 1961, 1971, 1981 and 1991) and proxies for emissions (e.g. land cover, road traffic, industrial emission sources), information on environmental factors that might influence dispersion of the air pollutants (e.g. topography, meteorology) and information on population distribution and population characteristics that might act to determine exposures to ambient air pollution. Additional historical datasets identified for use in modelling include lichen surveys and smoke control areas (related to domestic fuel use).

Figure 8.7.1 Map of ordinary kriging annual black smoke concentrations for 1962



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Conclusions

The ‘best performing’ methods for each decade will be selected and used to produce small area (1 km grid where possible) air pollution maps of GB for each of the target years to develop individual exposure estimates for participants in the UK longitudinal survey, and potential for interpolating to intervening years will be assessed. In conclusion, kriging was found to provide reasonable ‘benchmark’ maps against which the further GIS ‘smart’ modelling (with covariates) methodologies can be compared. A range of datasets including air pollution concentrations are available to aid modelling of historical exposures to air pollution in GB.

8.8 Nanoparticles in urban air: A very small problem

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Background

The human body is exposed to atmospheric pollutants on a daily basis. Breathing is the most effective route of airborne contaminant entry into the human body (BéruBé *et al*., 2008), meaning our lungs and airways take the full force of this exposure. Recent pollutant research has focused on how the smallest particles appear to be responsible for the greatest health effects, with the main focus placed on nanoparticles. Nanoparticles are defined as particles with at least one dimension under 100 nm (BéruBé *et al*., 2008). In order to put this into a biological perspective, the smallest human virus is 20 nm in diameter and is able to translocate freely throughout the body. In parallel, nanoparticles smaller than 20 nm (which are commonly found in urban air; Shi *et al*., 2001) could have the same translocation and deposition potentials; biological factors that currently remain unknown (Gwinn & Vallyathan, 2006).

Aims and objectives

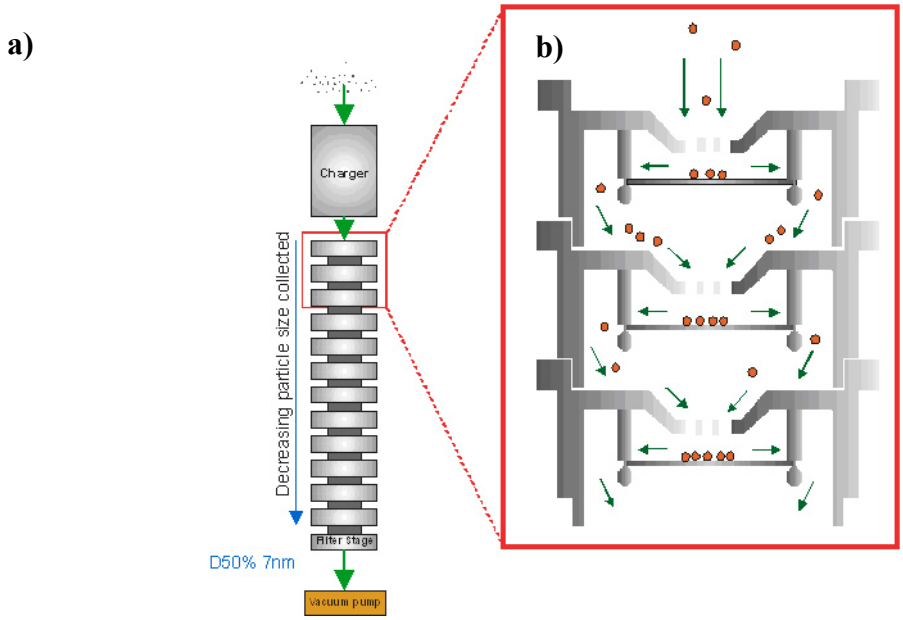
This study aims to investigate the physicochemical properties of nanoparticles in urban air, and relate these to their impact upon human health. The research objectives are three-fold and include:

- real-time measurement and actual particle collection of urban and background nanoparticles using the Dekati Electrical Low Pressure Impactor (ELPI)^{TM}
- physicochemical analysis of particles using high-resolution scanning/transmission electron microscopies and correlative microanalysis and mass spectrometry
- analysis of respiratory health impacts (e.g. inflammation, oedema and secretion) by testing the nanoparticles in a 3-dimensional, organotypic, *in vitro* model of human respiratory epithelia.

Methodology

The size of nanoparticles does not make them conducive for collection (SCENIHR, 2006), and their enhanced chemical and physical properties, when compared to similar particle types with a larger diameter (Seaton *et al.*, 1995), have caused significant sampling problems. However, recent equipment developments coupled with increasing public health concerns has resulted in a new generation of particle samplers. The Dekati ELPI (Figure 8.8.1; Keskinen *et al*., 1992) is an example of equipment specifically designed for nanoparticle sampling. It is designed to give both real-time measurements of ambient particle concentrations as well as trapping the measured particles onto collection substrates. The nanoparticles can then be micro-analysed *in-situ* on the substrates or extracted for *in vitro* analysis. The ELPI has been used to sample ambient, urban particles in a traffic pollution corridor (Neath Road), Swansea, South Wales from October 2007 to the present.

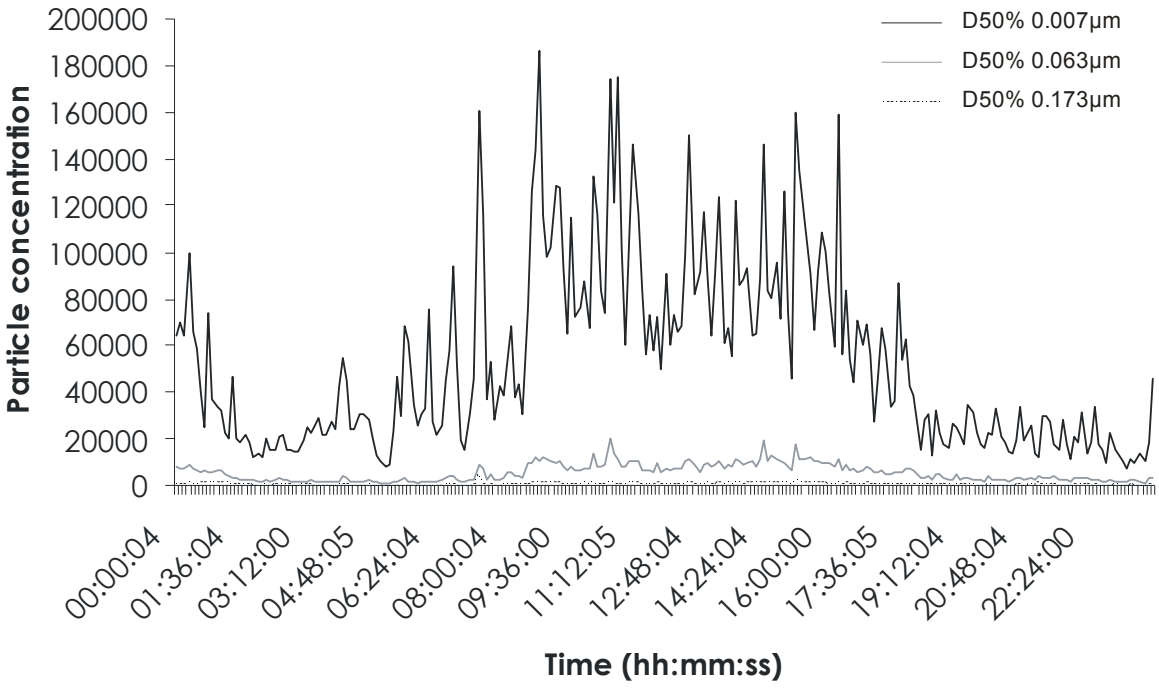
Figure 8.8.1 (a) The ELPI; (b) schematic diagram of the collection stages in the ELPI



Real-time measurements

Particles numbers, over a series of size ranges, were recorded every five seconds, which could then be averaged or processed as required. For example, particle number data could be compared according to time of day or week, season, meteorological conditions or vehicle numbers (Figure 8.8.2).

Figure 8.8.2 Comparison of particle sizes in a traffic corridor in Swansea, South Wales for three size fractions over 24 hours (11/01/2008) with a six-minute average



The Swansea traffic corridor contains particle numbers that vary in response to their size (Figure 8.8.2). The highest concentrations of particles were observed in the smallest size fraction (D50% 0.007 µm, where 50% of the particles have an average diameter of 7 nm). The next measured size fraction (D50% 0.063 µm) has a significantly lower particle concentration, and the D50% 0.173 µm

size fraction, a lower particle concentration again. These results corroborate those found by Roth *et al.* (2008), a study in which ultrafine particle numbers in air from Strasbourg, France were found to be consistently higher in concentration than the larger particles.

Particle collection

An advantage of the ELPI is the possibility of not only measuring real-time particle concentrations, but yielding actual particle samples which can then be analysed using a variety of morphological (electron microscopy and image analysis) and chemical (X-ray micro-analysis and mass spectrometry) techniques (Figures 8.8.3 and 8.8.4).

Figure 8.8.3 Field emission scanning electron microscopy (FESEM) image of a typical ELPI particle collection captured on a foil substrate, after one week sampling in Swansea, South Wales (D50% 0.063 µm). Particles are concentrated into individual particle 'piles' on the foil substrate

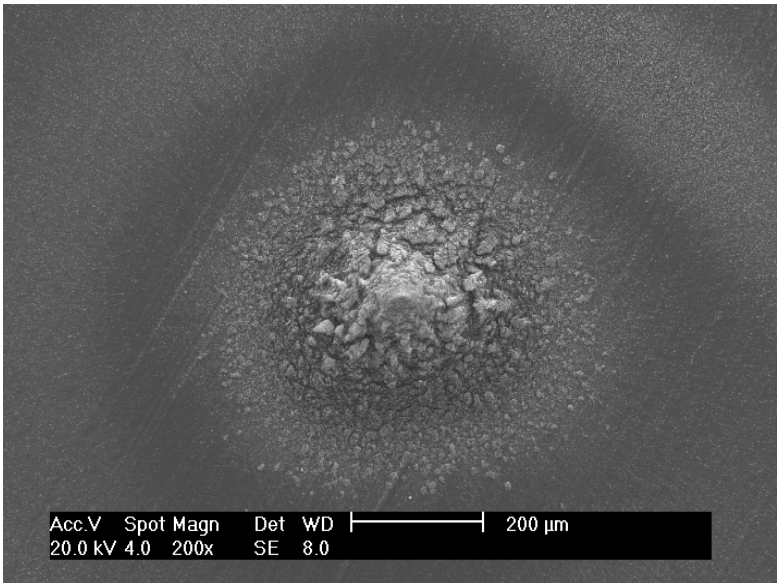
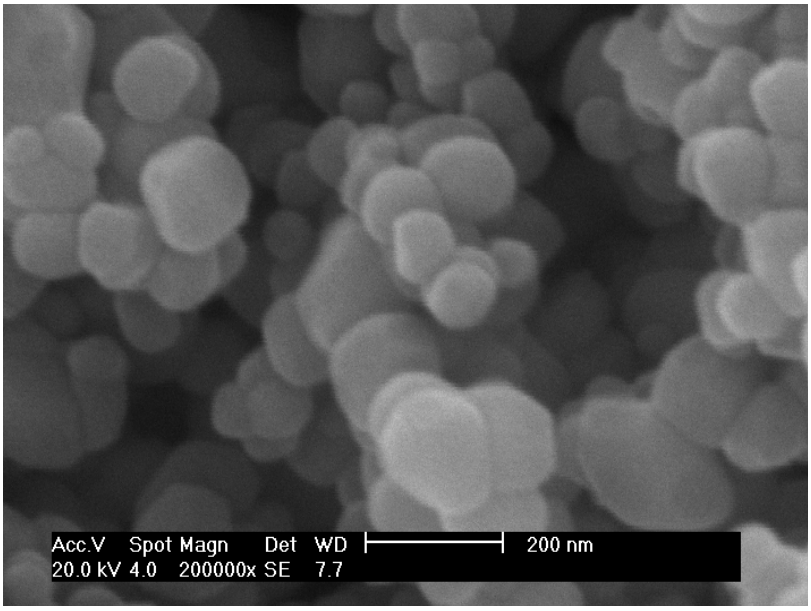


Figure **8.8.4** Higher magnification FESEM image of a particle 'pile'



Conclusions and future work

The ELPI has enabled the investigation of the dynamic activity of ambient aerosols in an urban traffic conurbation. The real-time capability of the collector, along with its capacity to trap particles on a substrate, has the added value of permitting the determination of correlative particle physicochemistry, factors that are lacking in nanoparticle toxicology studies.

Future work will include:

- Dekati ELPI equipment ground-testing focusing on the reliability of given particle numbers
- monitoring in a variety of urban locations and in all seasons
- physicochemical analysis of the collected particles
- analysis of the impact of urban air nanoparticles upon human tissue *in vitro*.

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8.9 Oxidative capacity of ambient PM_{10} collected at an industrial waste transfer facility

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Background and objectives

The highest PM_{10} concentrations in the London Air Quality Network (LAQN) have been measured at sampling sites in close proximity to waste transfer facilities (Fuller & Meston, 2008). Bexley 4 (BX4) is one example of this type of industrial site in the London Borough of Bexley. Located along Manor Road, Erith, this kerbside site is situated 30 m west of metal reclamation and waste transfer facilities and is also in close proximity to a residential community. BX4 has consistently breached the EU limit value/ air quality strategy objective for particulate matter with an aerodynamic diameter less than 10 μm (PM_{10}), stating the daily PM_{10} mean may only exceed a concentration of 50μg m^{-3} on a maximum of 35 days annually. Despite being declared an air quality management area in 2001, PM_{10} concentrations measured at BX4 have subsequently increased: the EU limit value was exceeded on 77 days and 116 days in 2001 and 2006, respectively (Fuller, 2008).

The majority of locally generated PM_{10} at BX4 has been primary attributed to vehicular non-tail pipe emissions, likely from road dust resuspension and dust being lifted directly from dirty vehicles (Fuller & Baker, 2001). Consequently studies reported the high PM_{10} concentration measured at this site was not a health concern but rather a local dust issue (ERM, 2006). However, this conclusion was based on the assumption that the compositional profile of BX4-sampled PM_{10} was biologically inert and was not supported by a toxicological assessment.

The objectives of this study were three-fold.

- To quantify the toxicity, measured as oxidative potential, of bulk daily PM_{10} BX4 exposures
- To evaluate PM_{10} oxidative activity as a function of the weekly industrial waste transfer facility emission cycle
- To analyse the influence of wind direction on the sampled particles and associated toxicological parameters

Methods

PM sampling

PM_{10} samples were collected daily on Teflon filters using the Automated Cartridge Collection Unit (ACCU) installed on the Tapered Element Oscillating Microbalance (TEOM). To increase filter mass loading, filters were exposed repeatedly between two and five times, but only on the same day of week as previous collections (i.e. all Monday sampling occurred on the same filter). This sampling methodology was conducted at Bexley 4 between 5 June and 30August, 2002.

Assessment of oxidative activity

The capacity of PM to induce oxidative stress was assessed *in vitro* by measuring antioxidant depletion in a synthetic human respiratory track lining fluid (RTLF) containing physiologically relevant concentrations (200 μM) of ascorbate (AA), urate (UA) and reduced glutathione (GSH). The RTLF model was exposed to equal masses of PM samples (50 μg mL^{-1}) for a period of four hours at 37 °C. Performing these experiments at equal doses enabled a comparison of the relative particle toxicity and elucidated whether infringements of the air quality directives at BX4 were likely to pose a significant additional health risk. Particle-free and known particle controls were run in parallel with

filter samples to ensure inter-experiment standardisation. Antioxidant concentrations were quantified following exposures.

PM metal composition

The influence of PM_{10} transition metal content on oxidative activity was evaluated using three methods.

- PM_{10} total metal concentrations (Al, As, Cd, Cu, Fe, Pb, Mg, Pt, W and Zn) were measured by ICP-MS.
- Bioavailable total and ferric iron concentrations in PM suspensions were quantified using the chromogenic chelator bathophenantroline disulphonate (BPS).
- The contribution of copper and iron specifically to PM oxidative activity was determined by incubating the PM samples with RTLF solution with and without the presence of a Cu^{2+} and Fe^{3+} transition metal chelator: diethylenetriamine-pentaacetic acid (DTPA).

Semi-continuous measurements

Semi-continuous TEOM PM_{10} and meteorological (wind speed, wind direction, temperature) measurements were made with 15-minute resolution.

PM_{10} source apportionment

PM_{10} concentrations were fractionated into three source components over the campaign period (1 June to 31August 2002): background natural and secondary; background primary; and BX4 local. The latter PM_{10} estimates were then used to quantify the fraction of PM_{10} measured at BX4 related to local activities.

Results and discussion

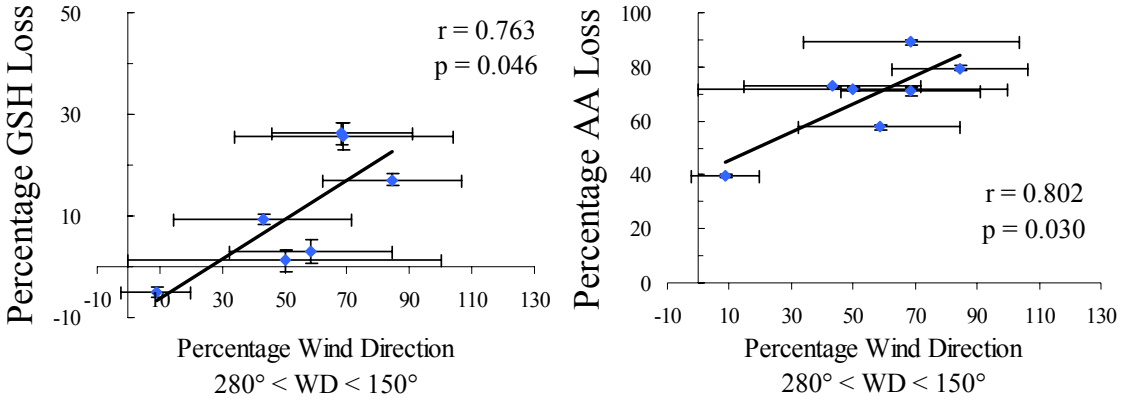
Locally generated PM_{10} exhibited a clear diurnal trend on weekdays, achieving a maximum during normal working hours. The concentration of this local PM_{10} fraction was highly sensitive to wind direction: between 280–150° the sampling site was predominately exposed to emissions from industrial facilities and the roadway. During periods of industrial emission exposure (85 μg m^{-3} maximum hourly averaged PM_{10} concentration), total PM_{10} concentrations significantly increased compared with residential emission exposure (24 μg m^{-3} minimum hourly averaged PM_{10} concentration); the cause for increased total PM_{10} was due to elevated local PM_{10} fraction concentrations.

The degree of ascorbate and reduced glutathione depletion relative to particle-free controls seen after incubation for each PM sample was a direct index of the oxidant activity of the particles in that sample. BX4 samples exhibited significantly greater PM oxidative activity on weekdays per unit mass, with a decline in PM reactivity on Saturday and the lowest activity observed in the Sunday sample. Although the highest antioxidant depletion was observed on weekdays, considerable variation existed within Monday to Friday samples: ascorbate losses ranged from 71 to 89% and reduced glutathione losses ranged from 1 to 26%. Similar weekday/weekend and within weekday variation trends were observed for total and ferric bioavailable iron and total metal concentrations.

Weekday industrial waste transfer facility emissions all exhibited an equivalent diurnal profile and maintained an approximately constant magnitude; thus, the resulting oxidative capacity of PM was not expected to significantly fluctuate from Monday to Friday. However, the observed variability in PM toxicity (comprising antioxidant depletion, the bioavailable iron pool, and total metal concentrations measurements) thus suggested that regardless of proximity, BX4 was not continually exposed to waste transfer emission. This was likely due to oscillations in wind direction. As a result, exposure scenarios were defined for each day of the week during the sampling period to account for the percentage of time sampled PM_{10} was influenced by industrial waste transfer site emissions.

Fifteen-minute averaged wind direction measurements were considered for each individual day included in the bulk filter exposures where wind direction measurements between 280° and 150° indicated sampled PM_{10} was influenced by industrial sources, while remaining bearings were classified as residential-derived PM_{10}. It was observed that days of the week predominantly influenced by industrial emissions corresponded with the highest levels of antioxidant depletion (Figure 8.9.1).

Figure 8.9.1 Pearson correlations between the percentage loss of reduced glutathione (GSH) and ascorbate (AA) relative to particle-free control samples and the percentage of time during each exposure period industrial waste transfer facility derived PM_{10} was sampled (predicted by a wind direction measured between 280° and 150°); n = 7



ICP-MS analysis revealed significant associations between the degree of ascorbate loss and total Pb (r = 0.882, p = 0.009) and Fe (r = 0.797, p = 0.032) metal concentrations. In contrast, the loss of reduced glutathione was not related to Fe, but rather to Al (r = 0.759, p = 0.048) and Pb (r = 0.888, p = 0.008) concentrations. Furthermore, Pb was strongly correlated to both Fe and Al. In addition, Pb was influenced by wind direction: significantly higher concentrations were found when sampled emissions originated from local industrial facilities.

Conclusions

PM_{10} oxidative activity

The PM_{10} local fraction was elevated during the day on each weekday and in the morning on Saturdays. However, on Sundays the local other fraction was reduced and no local primary PM_{10} fraction was detected. This study was designed to utilise this known pattern of locally produced PM_{10} at BX4 and compare the oxidative activity of daily sampled PM_{10}. A single filter was exposed repeatedly to ensure adequate PM mass availability for oxidant and metal analysis. A distinct difference was expected between weekday and Sunday samples, with filters exposed on Saturdays lying between the two. Although this general trend was observed, considerable variability between weekday sample oxidant activities was measured**.** The extent to which industrial emissions influenced sampled PM_{10} concentration, as predicted by wind direction, was used to explain this weekday variation. Moreover, elevated PM_{10} oxidative activity was established when local industrial waste transfer facility emissions predominately contributed to particulate PM_{10} sampled at BX4. Additionally, it is likely the measured PM toxicity was attributable to the compositional profile of the local other and local primary PM_{10} fractions, given their high sensitivity to wind direction**.**

PM_{10} composition

Oxidant activity induced by waste transfer facility emissions was compared to total metal and bioavailable iron PM content. Correlations between total transition metals and antioxidant depletion

produced significant associations between metal species and reduced glutathione (Al, Pb) and ascorbate (Fe, Pb) depletion. Moreover, the concentration of Pb was strongly associated with both Fe and Al, suggesting that they have the same source(s). Total Fe and Pb exhibited a significant difference between the concentration on weekdays, Saturdays, and Sundays following the pattern of locally produced PM_{10} at BX4.

Assessing an overall health risk

PM_{10} infringements at BX4 were the result of industrial waste transfer facility-related emissions**.** Previous studies suggested the large amount of locally produced PM was the result of road dust resuspension from vehicular traffic along Manor Road servicing the local industrial facilities; ambient PM_{10} was thus postulated to be composed of biologically inert material thus yielding no health concerns. However, this study's detailed chemical characterisation and assessment of oxidative activity showed the high PM_{10} concentrations at BX4 were associated with elevated transition metal concentrations which consequently enhanced the ability of ambient PM to deplete antioxidants. Thus, PM_{10} generated by the waste transfer facility at BX4 should be considered a potential health risk to the neighbouring residential community.

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8.10 Bioreactivity of airborne PM_{10} particulate matter from a municipal solid waste landfill

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Background

With an increasing population and greater pressure on land use, the issue of landfilling is unavoidable and urgent. There are strong national and international concerns about the possible adverse health effects arising from living in the vicinity of municipal waste landfills: 80% of the UK population now reside within 2 km of a waste site. An understanding of the range of landfill emissions' toxicity is vital in determining the potential effects these could have upon nearby populations and the surrounding environment. In the past, landfill emissions in the form of gas and leachate have been the subjects of various investigations (ATSDR, 2002). However, significantly less information exists on landfill airborne particulate matter under 10 μm equivalent spherical diameter (PM_{10}). This investigation aims to determine the levels of landfill particulate air pollution and quantify the associated risks to the surrounding communities.

Aims

- Collect PM_{10} fractions during the summer months from an active municipal landfill site and an urban location
- Field emission scanning electron microscopy (FESEM) for morphological characterisation
- Screen for bioreactivity using the ROS-sensitive plasmid scission assay

Methods

Sample collection

The landfill of interest is a municipal solid waste landraise, located approximately 3.5 km from the centre of Cardiff, UK. A PM_{10} collection at Cardiff University's School of Biosciences was also conducted for urban comparison. The landfill site lies to the east of Cardiff city centre, situated on flat-lying ground known as the Wentlooge Levels. It is bounded to the north by a busy road, to the south by the Severn Estuary tidal mudflats, to the west by the mouth of the Rhymney River and to the east by a drainage ditch. The urban sampling site is approximately 500m from Cardiff city centre, with busy roads surrounding the location.

Sampling was undertaken during the summer of 2007. Two collecting systems were utilised in this study. A Negretti sampler was used to collect PM_{10} on polycarbonate filters for morphological characterisation by FESEM and image analysis. A high-volume collector configured to collect 10–2.5μm and <2.5μm was used to accumulate PM_{10} on polyurethane foam (PUF) filters. Due to logistical considerations and the inherent changing nature of an operating landfill site, landfill samples were collected during the peak operational hours of the site, while Cardiff city samples were collected for 10-hour periods. Toxicity screening was by the ROS-sensitive plasmid scission assay (PSA; Donaldson *et al.*, 1997), with the oxidative capacity of the particulate (PM) confirmed by EDTA-metal chelation.

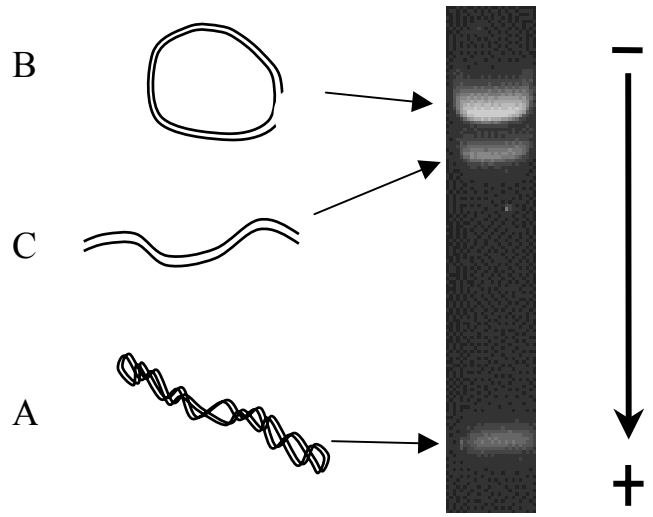
Characterisation

The polycarbonate filters were cut into triangular sections and mounted on metal-free plastic drums glued to SEM pin stubs. These were gold coated (SC500 Biorad sputter coater) before being viewed under FESEM (Philips XL30; secondary electron mode; accelerating voltage 20kV; working distance 5–10 mm, spot size 4). Quantitative image analysis utilised standard routines (Leica Q500IW imaging workstation, previously described in BéruBé *et al*., 2003).

Bioreactivity

Particles were extracted off the PUF filters using HPLC-grade water, and these $PM_{2.5}$ and $PM_{10–2.5}$ extracts were freeze-dried before being resusupended in molecular biology grade water. A proportionally representative combination of the two fractions was generated, and labelled as PM_{10}-whole. All unused stock PM solutions were stored at –80˚C. The *in vitro* toxicity system, known as the plasmid scission assay (PSA) has been widely used by researchers to evaluate the oxidative capacity of airborne PM (Moreno *et al*., 2004), and has been described in detail in Koshy *et al.*, 2007. In brief, commercially sourced ΦX174 RF plasmid DNA was incubated with a suspension of PM_{10} at known concentrations for 6h (n = 4–6), and the ensuing three different plasmid conformations were separated by gel electrophoresis. The extent of damage to the plasmid DNA (linearised plus relaxed forms) was reported as a percentage of the total amount of DNA present (Figure 8.10.1). The two different fractions obtained from the high-volume collector were independently assessed for toxicity in the PSA, along with a representative PM_{10}. In order to confirm the potential of the PM to generate ROS, the PSA was adapted to include a final concentration of 10 μM EDTA with the PM_{10} suspension. Statistical analyses included the Anderson–Darling normality test, the Student's t-test for significance in Minitab 14, and Graphpad Prism 2 for regression analysis.

Figure 8.10.1 An example of the plasmid scission assay, depicting the three different forms of post-exposure ΦX174 RF plasmid DNA. The post-incubated DNA is separated into these forms by agarose gel electrophoresis



A, undamaged, supercoiled form; B, nicked plasmid (moderate) damage; C, linearised plasmid (severe) damage

Results

Characterisation

Gravimetric analysis of landfill and urban PM_{10} showed similar size distribution of PM_{10} at these two locations; however it also revealed significantly increased PM_{10} generation at the landfill compared to the urban city location. The average mass concentration of the landfill sample was greater than that of the Cardiff sample (Table 8.10.1). Both locations generated PM_{10} with the majority of particles within the $PM_{2.5}$ range. The landfill PM_{10} contained a slightly wider size distribution within the PM_{10} fraction when compared to the Cardiff samples (Figure 8.10.2). Source apportionment of the urban collection revealed that both landfill PM_{10} and Cardiff PM_{10} were largely derived from anthropogenic sources (soot particles), with little evidence of biological matter (Table 8.10.2; Figure 8.10.3).

Table 8.10.1 Gravimetric evaluation of Cardiff municipal landfill and urban PM_{10} Summer 2007 collections, revealing the much higher mass concentration at the landfill compared to the urban site

Location	Duration (h)	Total mass (mg)	Average PM_{10} mass ($\mu g/m^3$)
Municipal landfill	10.75	32.7	42
Cardiff City (urban)	41	28.7	29

Table 8.10.2 Source apportionment of municipal landfill and Cardiff (urban) PM_{10} collections from summer 2007, as a percentage of total particle number. Note that although anthropogenic PM dominates at both sites, the municipal site PM_{10} has a much larger mineral content than the urban collection site

Location	Anthropogenic %	Mineral %	Biological %
Municipal landfill	71	29	0
Cardiff City (urban)	95	4	1

Figure 8.10.2 Size distribution of Cardiff urban and landfill PM_{10} Negretti collection. The vast majority of PM_{10} from both locations are composed of PM less than 2.5 µm ESD

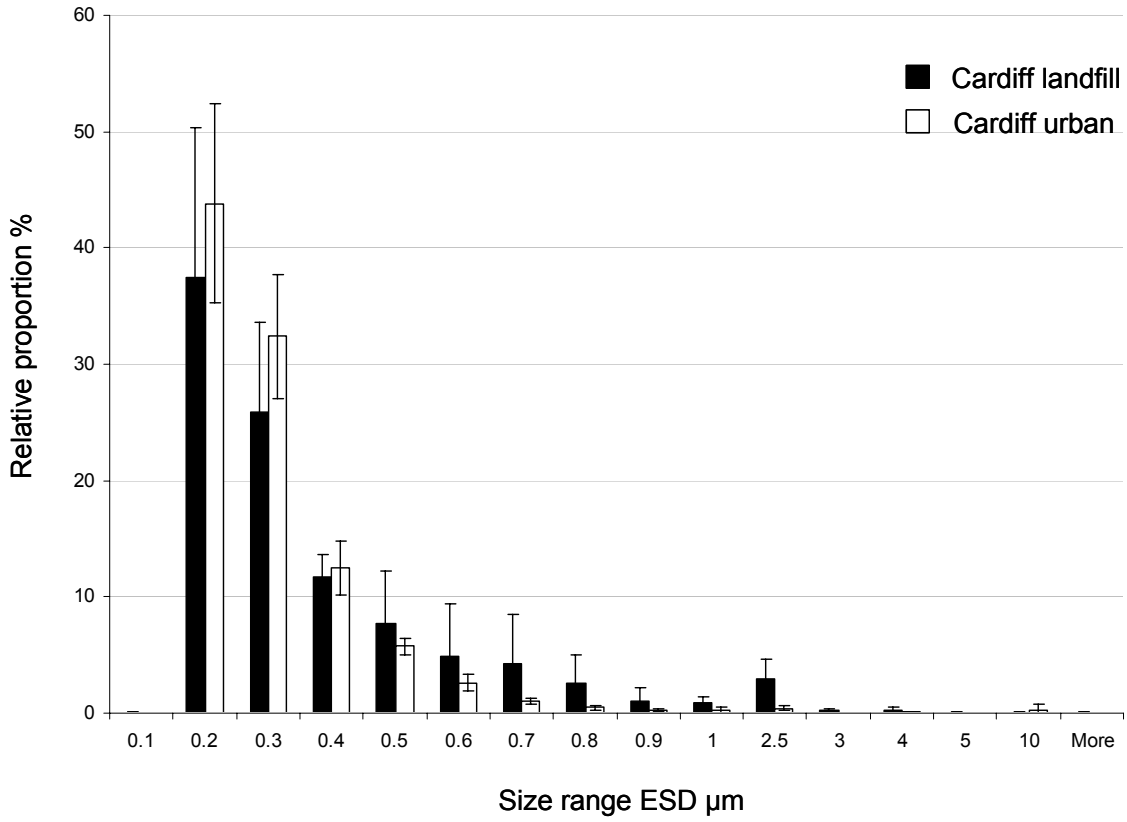
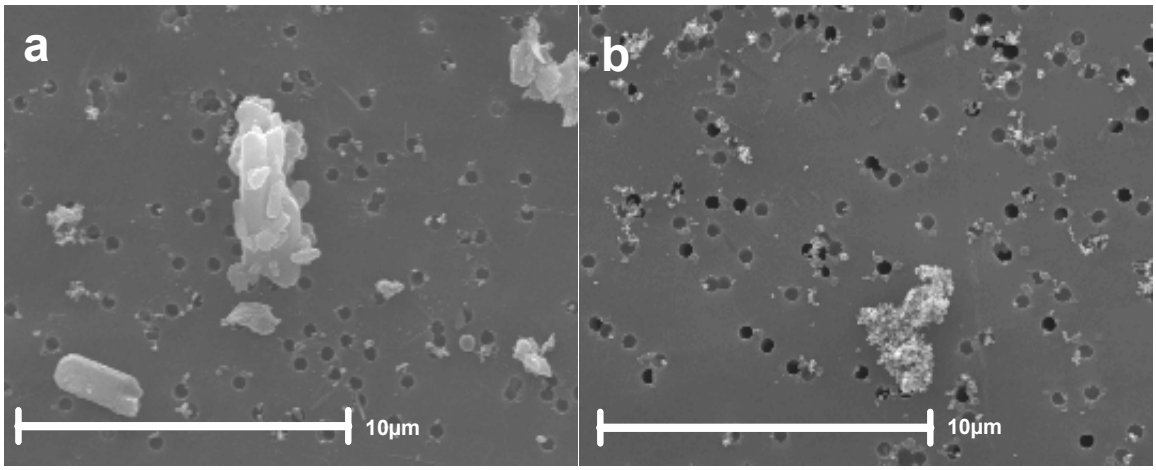


Figure 8.10.3 (a) PM_{10} collected at a Cardiff municipal landfill containing more mineral component than PM_{10} collected at the Cardiff urban location (b) PM_{10} collected at the Cardiff central urban location is mostly composed of anthropogenic, soot matter



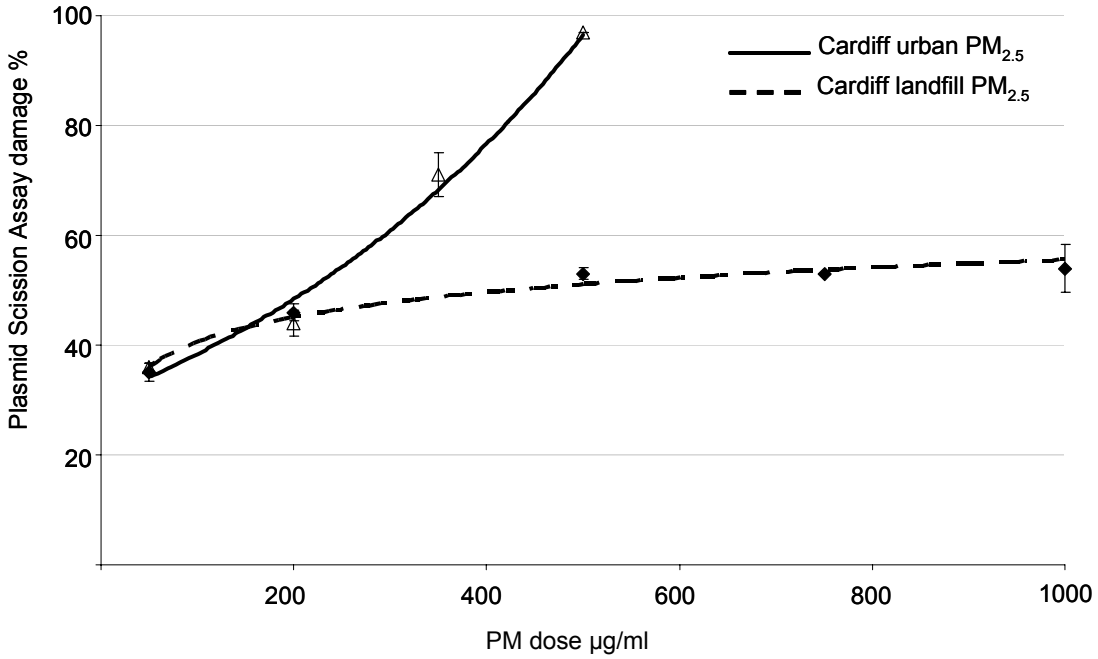
Bioreactivity

Overall, the bioreactivity of the landfill PM was found to be significantly less than that of the comparative urban PM collection, with little difference in plasmid DNA damage produced by the landfill $PM_{2.5}$, $PM_{10–2.5}$ or PM_{10}-whole fractions. The urban $PM_{2.5}$ fraction was significantly more bioreactive than either the $PM_{10–2.5}$ or representative PM_{10}-whole fractions, with 500 µg/ml $PM_{2.5}$ causing 97% damage in the PSA. The two PM collections exhibited different responses in the PSA from their respective $PM_{2.5}$ collections (Figure 8.10.4), with urban $PM_{2.5}$ (TD_{50} 185 µg/ml) adhering to an exponential response, while the equivalent landfill sample (TD_{50} 413 µg/ml) exhibited a hyperbolic response. Incubations of the reactive urban $PM_{2.5}$ fraction with EDTA caused a highly significant amelioration of damage ($p < 0.001$).

Conclusions

This study has provided preliminary data on the physical characterisation and *in vitro* bioreactivity of airborne municipal landfill PM_{10}, which was compared to an urban PM_{10} sample. Although the average mass concentration of the landfill PM_{10} was higher than that of the urban PM_{10} collection, these samples were collected during the peak operating hours of the waste site, and may not have been a genuine representation of the 24h average exposure. Greater bioreactivity in the PSA was observed with the urban PM_{10}, compared with the landfill PM_{10}, which may be due to the increased levels of combusted material present in urban locations. The highly significant reduction in bioreactivity caused by the addition of EDTA to incubating suspensions of the urban $PM_{2.5}$ supports the hypothesis that metals in urban respirable PM cause reactivity. Future work is now being undertaken to characterise the metal content of landfill and urban PM_{10} collections, and to assess the variability in PM_{10} generated from different locations of the landfill.

Figure 8.10.4 Hyperbolic and exponential dose–response curves observed in the plasmid scission assay of Cardiff municipal landfill and urban $PM_{2.5}$ collections. Note the urban sample is significantly more reactive than the equivalent landfill fraction, with calculated TD_{50} values of 185 and 413 µg/ml, respectively (regression analysis in Graphpad prism)



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8.11 Reactive oxygen species drives coal fly ash bioreactivity

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Background and objectives

The term 'fly ash' is used to describe particulate matter (PM) derived from mineral and metal contaminants within solid (e.g. coal) and liquid (e.g. oil) organic fuels (Jones & BéruBé, 2006; BéruBé *et al*., 2008). Coal fly ash (CFA) is one of these products of incomplete combustion, and is small and light enough to be released into the atmosphere. CFA particles are 60–90% glass and may contain many metals, such as Al, Fe, Ni, V, As, Be, Cd, Cu, Zn, Pb, Se, Rn and Mo (Donaldson *et al*., 2005; Jones and BéruBé, 2006). Additionally, these particles can contain recrystallised minerals, mainly quartz, mullite and haematite. Molecular toxicology studies have confirmed (BéruBé *et al*., 2007) that combustion-derived particles and their components may exert oxidative stress, which leads to activation of signaling pathways and stimulates pro-inflammatory gene expression. The aim of this study was to develop a better understanding of the potential bioreactivity of CFA because it includes a fraction of PM_{10} particles (i.e. particles with aerodynamic diameter less than 10 μm) which are recognised as a respiratory human health threat (BéruBé *et al*., 2007; BéruBé *et al*., 2008). To accomplish this, a multi-disciplinary approach was employed, using geological and biochemical methodology to characterise CFA derived from a Welsh power station.

Study description

Geological analysis of coal fly ash

The physicochemistry of the CFA particles was determined by field emission scanning electron microscopy (FE SEM) to study particle morphology, size and surface textures. X-ray diffraction (XRD) provided data on the type of crystalline mineral phases present. Inductively coupled plasma-mass spectroscopy (ICP-MS) provided information about the elemental composition. A respirable dust separator system was used to obtain PM_{10} samples from bulk FA collected from the top, middle, and bottom part of the CFA tip from the power station.

Biochemical analysis of coal fly ash bioreactivity

A DCFH (2',7'-dichlorodihydrofluorescin) assay was the method used to quantify the amount of reactive oxygen species (ROS) generated by 0.5, 1, 2, and 4 mg/ml solutions of FA samples. In the presence of ROS, non-fluorescent DCFH dye was oxidised to highly fluorescent DCF (Figure 8.11.1). The detected fluorescence was calibrated against an equivalent H_2O_2 concentration for a given probe. The plasmid scission assay (PSA) provides information about damage caused by CFA to plasmid DNA. The bacteriophage ΦX174 RF DNA (Figure 8.11.2) was used, as its super-coiled DNA becomes relaxed (damaged) and finally linear (severely damaged) in the presence of ROS generated by 0.5, 1 and 2 mg/ml solutions of CFA after 6 h incubation.

Figure 8.11.1 A schematic diagram of the DCFH assay depicting the oxidation of non-fluorescent DCFH dye to fluorescent DCF dye via ROS generated from CFA particles

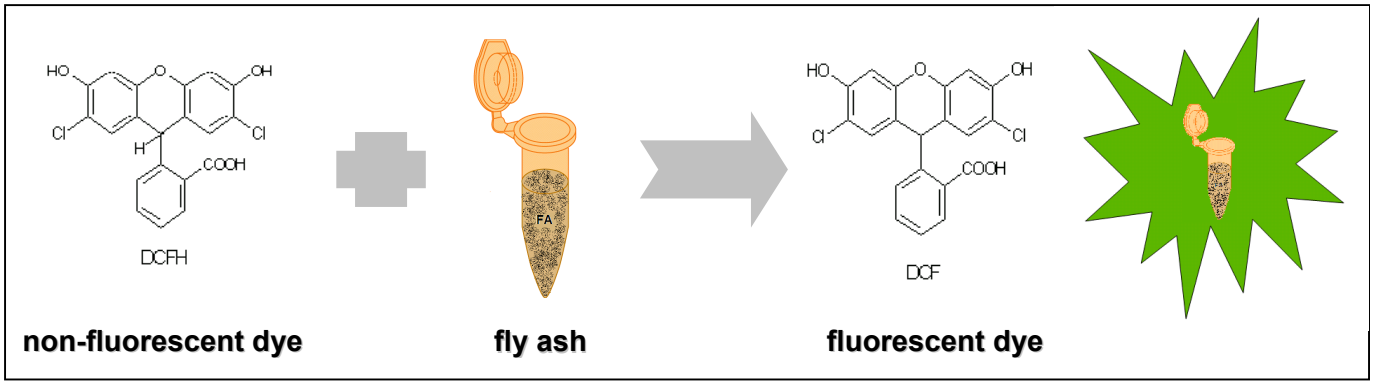
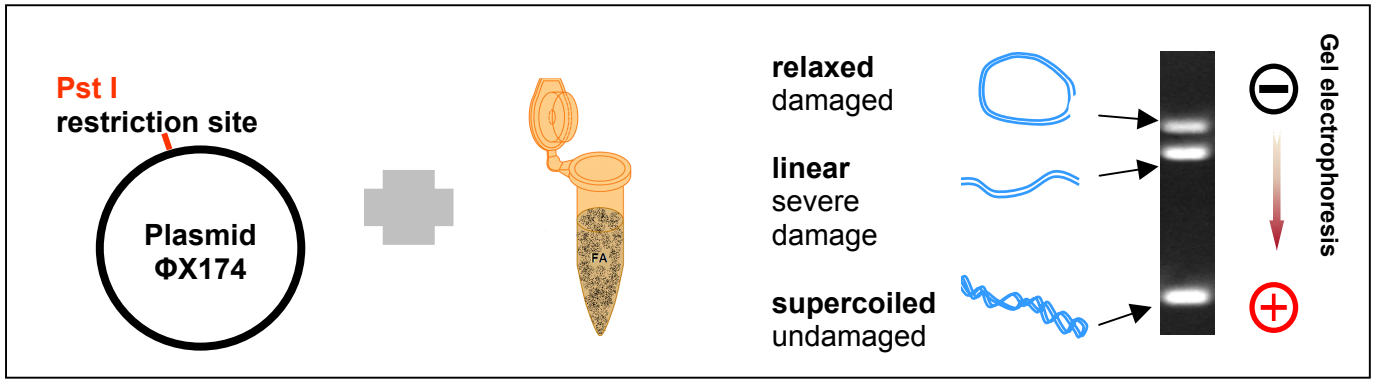


Figure 8.11.2 An overview of the plasmid scission assay demonstrating the morphological changes in bacteriophage ΦX174 RF super-coiled DNA in the presence of ROS produced by CFA: the morphologies include 'relaxed' (damaged) and 'linear' (severely damaged) forms



Discussion

Geological analysis

The FE SEM images show that the PM_{10} fraction of CFA generally possess a spherical shape (Figure 8.11.3), and approximately 80% of those spheres have a diameter between 0.1–2.5µm. XRD analysis confirmed the presence of crystalline phases such as quartz, mullite and haematite (Figure 8.11.3), and ICP-MS (data not shown) revealed that the elemental compositions were dominated by Si, Al, and Fe.

Biochemical analysis

The results from the biochemical assays demonstrated the ability of CFA to generate ROS, which was identified by the fluorescence intensity of DCF dye (Figure 8.11.4) or damage caused to plasmid DNA (Figure 8.11.5). For the DCFH assay, a 0.5mg/ml solution of CFA produced the highest level of oxidative capacity. The PSA data for the top, middle, and bottom samples of CFA were comparable, and did not show a dose-dependent response.

Conclusions

The combination of geological and biochemical analysis have shown that CFA is more than a nuisance environmental dust. It contains a fraction of PM_{10} particulate matter, most of which is fine

particles (0.1–2.5 µm). This fraction is known to be highly respirable and to deposit in the tracheobronchial region of the human respiratory tract. CFA contains a number of metals and crystalline mineral phases, amongst which quartz and mullite are recognised respiratory hazards. The DCFH and PSA data correlate well, and suggest that ROS is the driving force behind the CFA bioreactivity. Therefore, the small respirable size of these CFA particles, along with their oxidative capacity, would suggest that CFA should be considered a potentially hazardous particulate air pollutant.

Figure 8.11.3 An FE SEM image of CFA particles, and a respective X-ray diffraction pattern showing peaks for the minerals quartz, mullite and haematite; crystalline particles may present a risk of lung fibrosis

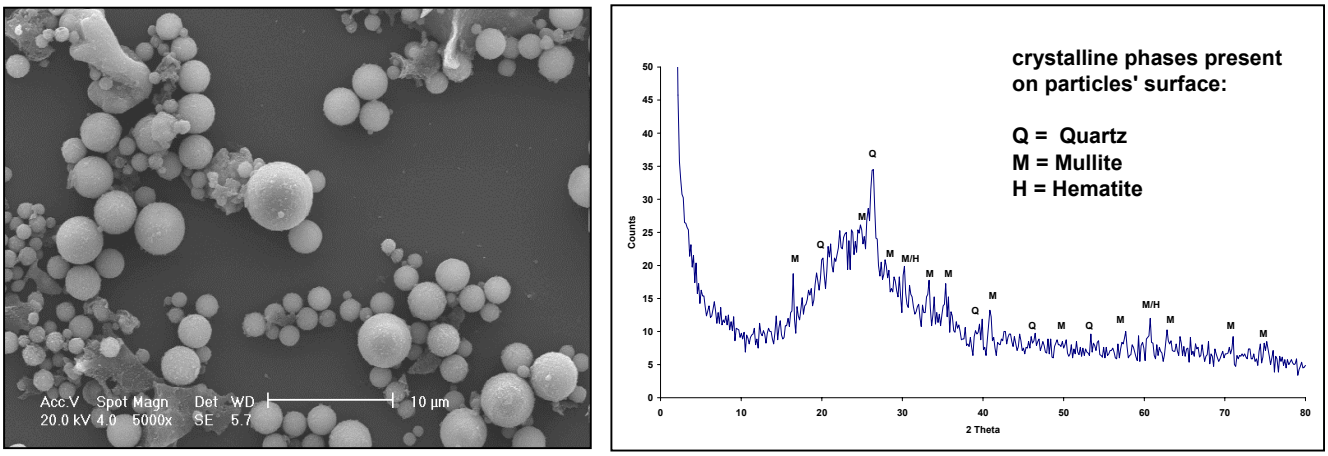


Figure 8.11.4 Reactive oxygen species generated by 0.5, 1, 2 and 4 mg/ml solutions of PM_{10} samples from the top, middle and bottom of the CFA pile. Data show a significantly higher amount of ROS generated by the top fraction of Aberthaw PM_{10} compared to middle and bottom fractions. Error bars = SD

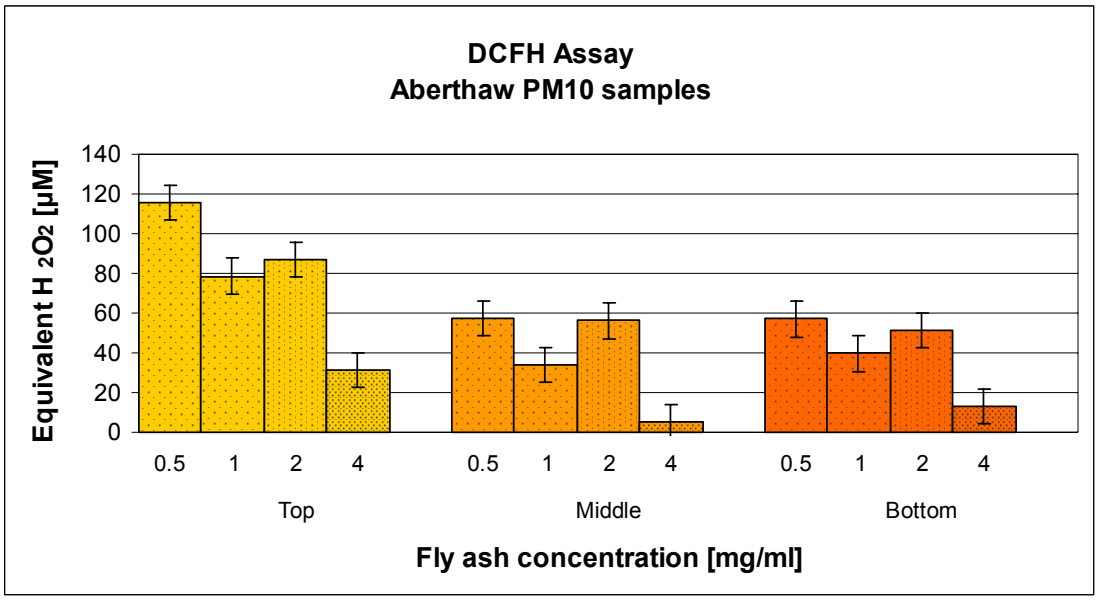
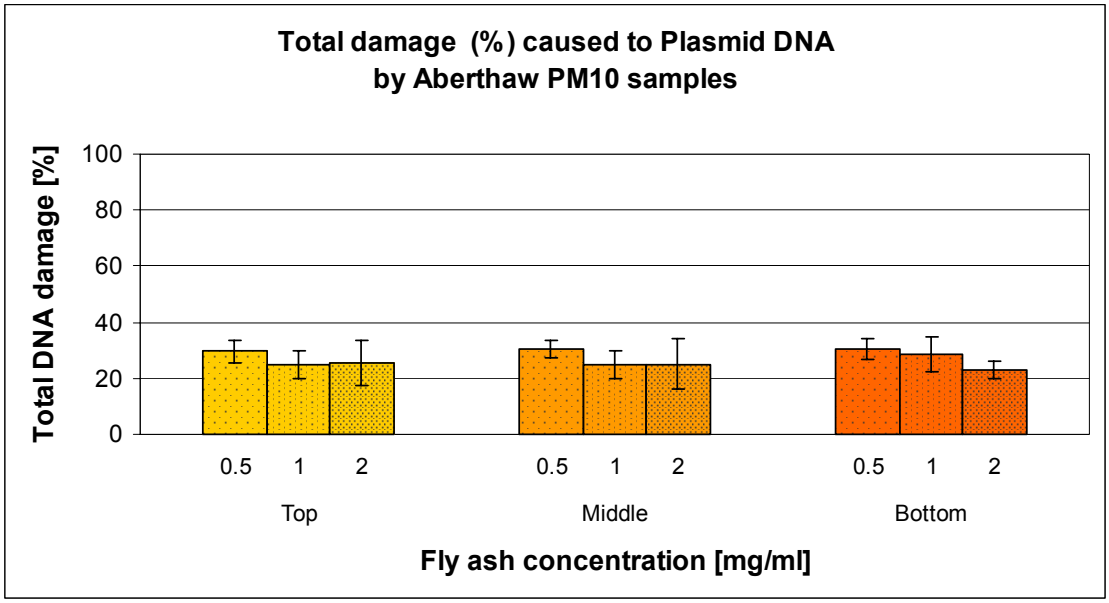


Figure 8.11.5 Damage to plasmid DNA caused by different concentrations (0.5, 1 and 2 mg/ml) of PM_{10} samples from the top, middle and bottom of the CFA pile. The relative amount of damaged DNA (relaxed and linear forms) was calculated as a percentage of the total DNA. Error bars = SD



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8.12 The effect of particulate air pollution on birth weight: Results from the UK PAMPER study

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Background and objectives

There is accumulating evidence to suggest that exposure to ambient air pollution has an adverse effect on birth weight and low birth weight (Glinianaia *et al*., 2004; Lacasana *et al*., 2005; Maisonet *et al*., 2004; Sram *et al.,* 2005). Studies of different designs such as ecologic (Bobak & Leon, 1999), case–control (Rogers *et al.*, 2000), cohort (Chen *et al.*, 2002; Wang *et al.*, 1997; Wilhelm & Ritz, 2003) or longitudinal follow-up cohorts (Jedrychowski *et al*., 2004) have consistently implicated air pollutants in causing adverse birth outcomes, but with varying risk magnitude. Currently such evidence is available from different countries across all continents except Africa. A range of exposure assessment methods, including arbitrary assignments of exposure based on geographical location (Yang *et al*., 2003), or proximity to different polluting sources such as roads (Wilhelm & Ritz, 2003), mathematical modelling (Rogers *et al.*, 2000), as well as personal monitoring (Jedrychowski *et al.*, 2004), have been considered in these studies. Evidence from studies with such a variety of study designs, different populations and exposure assessments suggests that the observed effect on birth weight and low birth weight is consistent.

Existing studies were based on few years, with less variability in the exposure range than in our study. Most of the previous studies used exposure as a categorical variable (quartiles, percentiles etc.) that limited the scope of the analysis to step change in effect. In this study, that covers 32 years, we investigated the effect of black smoke air pollution on birth weight. The considerable variability in the exposure range increased the power of the study to detect any significant associations.

Study description

These analyses are part of the historical cohort UK PAMPER (Particulate Matter and Perinatal Events Research) study investigating an association between maternal exposure to black smoke air pollution and adverse perinatal outcomes. Using data from paper-based neonatal records from the two major maternity hospitals in Newcastle upon Tyne, we constructed a birth record database of all singletons born during 1961–1992 (N = 109 086) to mothers resident in the city. The PAMPER database contained information on birth weight, gestational age, infant gender, maternal age, parity and neighbourhood socio-economic status. Weekly black smoke exposure estimates for each individual pregnancy were derived from a two-stage modelling process incorporating monitored black smoke data with temperature and pollution source information. The modelling approach is described in detail elsewhere (Fanshawe *et al*., 2007) and was presented at the Tenth Annual UK Review Meeting on Outdoor and Indoor Air Pollution in 2007 (IEH, 2007).

We first estimated the crude association between black smoke and continuous birth weight by fitting a simple linear regression model, and subsequently adjusted for the above variables in a multivariable model. Following this step, we used fractional polynomials to check for any evidence of non-linear relationships. We found non-linear models for the whole pregnancy and second trimester exposure models to be significantly better than the linear ones. The best fitting fractional polynomial model for whole pregnancy exposure was the reciprocal of the square root and the product of the reciprocal of square root and $\log\left\{\beta\left(1/\sqrt{x}\right)+\gamma\left(1/\sqrt{x}\right)\times\log(x)\right\}$, where x is whole pregnancy average black smoke. For the second trimester the best fitting functional form was the square root $(\beta\times\sqrt{x})$, where x denotes second trimester average black smoke. Unlike a linear relationship where the association is

the same for a unit change in exposure throughout the exposure scale, in a non-linear relationship it is different at different points on the exposure scale. We have chosen to report the associations at different standard points, for example, 1st, 25th, 50th, 75th and the 90th percentiles of exposures.

For the first and the third trimester exposure models there was no evidence ($p > 0.05$) that a non-linear model significantly improves the fit, therefore the linear models were considered adequate.

Results

Mean birth weight increased over the three decades. Mean birth weight was the highest in the highest quintile of the Townsend score, which is assumed to be the most affluent group. Most favourable birth weight was observed in the maternal age categories 25–29 and 30–34 years. Female infants had lower mean birth weight and higher percentage of low birth weight than males.

Four black smoke exposure estimates were considered in the current analysis: averages over the whole pregnancy and by each of the three trimesters. The whole pregnancy ranges from the date of conception to birth, first trimester ranges from the first week after conception to the 12th week, second trimester from the 13th to the 27th week, and the third from the 28th week until birth. Median exposures and the inter-quartile ranges were comparable for the four exposure windows; for the whole pregnancy it was 33.8 $\mu g/m^3$ (17.2, 108.3), for the first trimester it was 32.4 $\mu g/m^3$ (16.5, 94.2), for the second trimester it was 31.6 $\mu g/m^3$ (16.5, 94.9) and for the third trimester it was 30.1 $\mu g/m^3$ (15.9, 91.0).

Black smoke and birth weight

Whole pregnancy exposure

The unadjusted estimate showed a 3.3 g (95% CI: 2.8–3.8) decrease in birth weight for every 10 $\mu g/m^3$ increase in whole pregnancy black smoke exposure. The estimate reduced to 2.2 g (95% CI: 1.4–3.0) after adjusting for gestational age, Townsend deprivation score quintiles, maternal age, gender, parity and year of birth. Following this, we checked for any evidence of a non-linear relationship between birth weight and whole pregnancy exposure. A non-linear model was found to be significantly better ($p = 0.043$) than the linear model, suggesting a non-linear relationship between black smoke and birth weight. The best fitting non-linear model (see Study description above) showed that for an increase in exposure from the 1st (7.4 $\mu g/m^3$) to the 25th percentile (17.2 $\mu g/m^3$), predicted birth weight reduced by 3.6 g adjusted for all the covariates mentioned above. An increase in exposure from the 1st to 50th percentile (33.8 $\mu g/m^3$) reduced estimated birth weight by 24.0 g. A similar increase in exposure from the 1st to the 75th (108.3 $\mu g/m^3$) and 90th percentile (180.8 $\mu g/m^3$) birth weight is estimated to decrease by 69.6 g and 89.3 g respectively. When compared with the linear model the estimates from the non-linear model were higher for similar increases in exposure.

First trimester exposure

The unadjusted estimate shows that birth weight reduced by 2.0 g (95% CI: 1.6–2.4) for each 10 $\mu g/m^3$ increase in weekly black smoke exposure averaged over the first trimester of pregnancy. After adjustment for the covariates (Townsend deprivation score quintiles, maternal age, gender, parity and year of birth) the estimate reduced to 0.3 g (95% CI: 0.2–0.8).

Second trimester exposure

The unadjusted estimate shows that birth weight reduced by 2.7 g (95% CI: 2.2–3.1) for each 10 $\mu g/m^3$ increase in weekly black smoke exposure averaged over the second trimester of pregnancy, which reduced to 1.6 g (95% CI: 1.0–2.1) after adjustment for the covariates listed above. Further exploration of the non-linear relationship using fractional polynomials yielded a significantly better ($p = 0.022$) non-linear model. The adjusted model predicted a reduction in birth weight by 6.5 g when the exposure increased from the 1st percentile (6.3 $\mu g/m^3$) to the 25th percentile (16.5 $\mu g/m^3$). For an

increase from 1st to 50th (30.4 μg/m^3) percentile, estimated birth weight reduced by 13.0 g. The reduction for the 75th percentile (94.8 μg/m^3) and 90th percentile (186.7 μg/m^3) was 30.4 g and 46.9 g respectively.

Third trimester exposure

The unadjusted estimate for the third trimester exposure (weekly black smoke exposure averaged over the third trimester) was 2.9 g (95% CI: 2.5–3.3), slightly higher than that obtained for the first two trimesters. However, upon adjustment estimated birth weight was found to reduce to 0.9 g (95% CI: 0.4–1.5) for every 10 μg/m^3 increase in third trimester black smoke exposure.

Conclusion and discussion

This study, with its novel approach to exposure assessment and analyses, found an inverse association between black smoke particulate air pollution and birth weight and the relationship was non-linear. The estimated association observed in this study equates to a 70 g decrease in birth weight when black smoke exposure increased from about 8 μg/m^3 (1st percentile) to 108 μg/m^3 (75th percentile) for the whole pregnancy. The extent of the association observed at the lower end of the exposure scale did not increase at the same rate at the higher end of the exposure scale. For example, when the exposure increased from 8 μg/m^3 to 108 μg/m^3 estimated birth weight reduced by 70 g, but the estimated decrease was much less, about 25 g, when the exposure increased from 108 μg/m^3 to 208 μg/m^3. This demonstrates that the association was stronger at the lower end of the exposure scale and subsequent increase in exposure did not increase the association to the same extent. The results from this study are not directly comparable to similar ones conducted elsewhere because the pollutant black smoke has not been studied previously, nor have non-linear relationships been investigated except in the Polish study that reported a logarithmic relationship (Jedrychowski *et al*., 2004). Black smoke is considered to be approximately PM_4, which is between PM_{10} and $PM_{2.5}$, the most commonly investigated pollutant. Moreover, the non-linear relationship renders it incomparable to other studies that either used exposure categories such as quartiles or quintiles, or continuous but linear functional form for exposure. Comparisons of the estimates from the linear model shows a 2.2 g (95% CI: 1.4–3.0) reduction in birth weight for every 10 μg/m^3 increase in whole pregnancy exposure, which is within the range of estimates reported in the previous studies.

Comparison with previous research

A review of the studies on particulate air pollution and fetal health published in 2004 concluded that the evidence was compatible with either a small adverse effect of particulates on fetal growth or with no effect (Glinianaia *et al*., 2004), while a further review that included more recent studies and included gaseous pollutants as well, stated that there may be some evidence of a causal effect (Sram *et al.*, 2005). A study from the USA that investigated continuous birth weight reported an 11 g (95% CI: 2.3–19.8) reduction in birth weight for each 10 μg/m^3 increase in PM_{10} during the third trimester (Chen *et al*., 2002). A Polish study reported a 140 g reduction in birth weight for 10–50 μg/m^3 increase in $PM_{2.5}$ (Jedrychowski *et al.*, 2004) while another study from California reported a 35 g (95% CI: 12.0–58.6) decrease for those who had >18.4 μg/m^3 $PM_{2.5}$ exposure compared to <11.9 μg/m^3 in term infants (Parker *et al*., 2004). A Chinese study reported a 7 g (95% CI: 4.1–9.9) reduction in birth weight for each 100 μg/m^3 increase in total suspended particulates during the third trimester (Wang *et al.*, 1997). In Brazil, birth weight was reported to decrease by 14 g (95% CI: 0.4–27) for every 10 μg/m^3 increase in PM_{10} exposure during the first trimester (Gouveia *et al.*, 2004).

Acknowledgement

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8.13 Brominated flame retardants in dust: Implications for human exposure

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Background and objectives

Polybrominated diphenyl ethers (PBDE) have been used widely as flame retardants (FR) However, several jurisdictions have banned the marketing and use of the penta- and octa-BDE products, while the deca-BDE flame retardant formulation is the subject of ongoing EU risk assessment (BSEF, 2008). There has been much recent attention on the significance of ingestion of indoor dust as a pathway of human exposure to PBDEs and other brominated FRs, such as hexabromocyclododecanes (HBCD). Particular concern has been expressed for children (Jones-Otazo *et al*., 2005), with the existence of a causal link between dust and human body burdens suggested strongly by the correlation between PBDEs in household dust and human milk (Wu *et al.*, 2007).

Study description

Indoor dust samples were collected in the West Midlands and Basingstoke. Samples were all taken under normal room-use conditions, to reflect actual human exposures as far as possible. The following microenvironment categories were selected for study: homes (living rooms, halls, and bedrooms, n = 30), offices (n = 18) and cars (n = 20). Sampling was conducted according to a clearly defined standard protocol by one of the research team and described in detail elsewhere (Harrad *et al.*, 2008). Concentrations of tri-hexa-BDEs were determined at the University of Birmingham, with those of higher brominated PBDEs, decabromodiphenyl ethane (DBDPE) and 1,2-bis(2,4,6-tribromophenoxy)ethane (TBE) measured at the University of Antwerp. The analytical protocols are described in detail elsewhere (Harrad *et al.*, 2008).

Results

Table 8.13.1 summarises the concentrations of the target BFRs in dust samples taken from each microenvironment category studied. Concentrations of most target compounds are in the order: cars > offices ≥ homes. Statistical analysis (ANOVA) of log-transformed data reveals concentrations of PBDEs 47, 49, 66, 99, 100, 154, and Σtri-hexa-BDEs are significantly higher ($p < 0.05$) in cars than in homes. Concentrations of PBDEs 47, 99, 100, 154, and Σtri-hexa-BDEs in cars also exceeded significantly those in offices. There were no significant differences in concentrations between cars, offices and homes for any of the other target compounds. Of particular note are the highly elevated concentrations of BDE-209 found in dust from one car (2 600 000 ng g^{-1} = 0.26%), and two homes (0.22 and 0.14%). To the authors' knowledge these are the highest concentrations of BDE-209 in indoor dust reported anywhere.

We have estimated exposure to both Σtri-hexa-BDEs and BDE-209 as these represent respectively the predominant congeners present in the penta- and deca-BDE formulations, along with DBDPE and TBE. We have assumed 100% absorption of intake and used average adult and toddler dust ingestion figures of 20 and 50 mg day^{-1}, and high dust ingestion figures for adults and toddlers of 50 and 200 mg day^{-1} (Jones-Otazo *et al*., 2005). We have then estimated various dust ingestion exposure scenarios for homes, offices and cars separately, using 5th percentile, median, average and 95th percentile concentrations in the dust samples reported here. Overall dust ingestion exposure estimates are then calculated taking into account ingestion of dust in each of the relevant microenvironments. Dust ingestion is assumed to occur pro rata to typical activity patterns (i.e. for adults 72% home, 23.8% office, 4.2% car; for toddlers 95.8% home and 4.2% car). Table 8.13.2 summarises these

estimates of the exposure of adults and toddlers to Σtri-hexa-BDEs, BDE-209, DBDPE, and TBE via dust ingestion.

Table 8.13.1 Summary of concentrations of BFRs in dust samples from indoor microenvironments

Location	Congener #	209 (ng g^{-1})	Σtri-hexa-BDE[a] (ng g^{-1})	ΣBDE[b] (ng g^{-1})	DBDPE (ng g^{-1})	TBE (ng g^{-1})
Homes	Mean	260 000	77	260 000	270	120
n = 30[c]	σ_n	580 000	68	580 000	770	430
	Median	8100	46	8500	24	5.3
	Min	<dl	7.1	12	<dl	<dl
	Max	2 200 000	250	2 200 000	3400	1900
Offices	Mean	30 000	250	31 000	170	7.2
n = 18[d]	σ_n	67 000	310	67 000	220	9.4
	Median	6200	100	7400	99	<dl
	Min	620	16	790	<dl	<dl
	Max	280 000	1100	280 000	860	40
Cars	Mean	410 000	2300	340 000	400	7.7
n = 20[e]	σ_n	770 000	5700	720 000	900	7.5
	Median	100 000	190	570 00	100	<dl
	Min	12 000	54	140	<dl	<dl
	Max	2 600 000	22 000	2 600 000	2900	29

[a] sum of PBDEs 28, 47, 49, 66, 99, 100, 153 and 154
[b] sum of PBDEs 28, 47, 49, 66, 99, 100, 153, 154, 183, 196, 197, 203 and 209
[c] thirty samples analysed for tri-hexa-BDEs; eighteen samples analysed for tri-deca-BDEs, DBDPE, and TBE
[d] eighteen samples analysed for tri-hexa-BDEs; fifteen samples analysed for tri-deca-BDEs, DBDPE, and TBE
[e] twenty samples analysed for tri-hexa-BDEs; nine samples analysed for tri-deca-BDEs, DBDPE, and TBE

Conclusions

While contamination with DBDPE, TBE and those PBDEs associated primarily with the octa- and deca-BDE formulations is consistent between cars, homes, and offices, contamination with those PBDEs contained mainly in the penta-BDE formulation differs between microenvironment categories. These findings are consistent broadly with our previous study of PBDEs in indoor air in the West Midlands (Harrad *et al.*, 2006), which showed concentrations in offices of the PBDEs prevalent in the penta-BDE formulation to exceed significantly those in homes, and revealed concentrations of the same congeners in cars to be the highest of the microenvironment categories studied. Furthermore, they are in line with a Japanese study that found office dust to be more contaminated with PBDEs than household dust, although no indication was given on whether this difference was statistically significant (Suzuki *et al*., 2006). Our car dust data are also consistent with the only previously reported information on concentrations of PBDEs in car dust, consisting of two samples from the USA (Gearhart & Posselt, 2006). Our study demonstrates car interiors are substantially contaminated with PBDEs, and suggests further monitoring of PBDE contamination in cars is warranted.

Table 8.13.2 Summary of estimates of exposure[a] (ng day^{-1}) of UK adults and toddlers to BFRs via indoor dust ingestion

	Adult				**Toddler (6–24 months)**			
Intake (ng Σtri-hexa-BDEs day^{-1})	5th %ile	Median	Arithmetic mean	95th %ile	5th %ile	Median	Arithmetic mean	95th %ile
Mean dust ingestion	0.31	1.3	4.2	22	0.66	2.6	8.5	45
High dust ingestion	0.78	3.3	11	54	2.7	10	34	180
	Adult				**Toddler (6–24 months)**			
Intake (ng BDE-209 day^{-1})	5th %ile	Median	Arithmetic mean	95th %ile	5th %ile	Median	Arithmetic mean	95th %ile
Mean dust ingestion	28	233	4300	24000	44	610	14000	78000
High dust ingestion	71	580	11000	61000	170	2400	54000	310000
	Adult				**Toddler (6–24 months)**			
Intake (ng DBDPE day^{-1})	5th %ile	Median	Arithmetic mean	95th %ile	5th %ile	Median	Arithmetic mean	95th %ile
Mean dust ingestion	0.06	0.91	5	17	0.12	1.4	14	48
High dust ingestion	0.15	2.3	12	44	0.5	5.5	54	190
	Adult				**Toddler (6–24 months)**			
Intake (ng TBE day^{-1})	5th %ile	Median	Arithmetic mean	95th %ile	5th %ile	Median	Arithmetic mean	95th %ile
Mean dust ingestion	0.03	0.09	1.7	5.3	0.06	0.26	5.6	17
High dust ingestion	0.08	0.23	4.3	13	0.23	1	22	69

[a] See text for assumptions employed to derive exposure estimates

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8.14 Methodological approaches to examining joint effects of noise and air pollution on health outcomes in populations living near major airports: Study design

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Background

Living near a major airport can mean exposure to both noise and air pollution from aircraft and from the road traffic serving the airport. Recent studies have found an association between exposure to transport noise and cardiovascular disease. The Hypertension and Exposure to Noise near Airports (HYENA) study, a Europe-wide research project looking at the effects of noise on populations living near major airports, has found significant exposure–response relationships between exposure to aircraft noise at night for both genders and road traffic noise in the day for men and the risk of hypertension (Jarup *et al.,* 2008). Studies have also found an association between air pollution and an increased risk of cardiovascular morbidity and mortality (Hoek *et al.,* 2002). However, no published studies have assessed the joint effects of noise and air pollution on health outcomes.

This is a complex issue to address because air pollution and noise often emanate from the same source. (Figure 8.14.1 below shows models of noise and air pollution from road traffic in the area to the east of Heathrow.) There are, however, some spatial differences in the distribution of the two pollutants in the vicinity of airports, since aircraft are a major source of noise pollution but not of air pollution. Road traffic, on the other hand, is a major source of both air pollution and noise. However, individual exposure to road traffic noise may vary substantially depending on, for example, window type, house insulation and bedroom location, whereas these modifiers of noise exposure may have less impact on the exposure to air pollution.

Aim

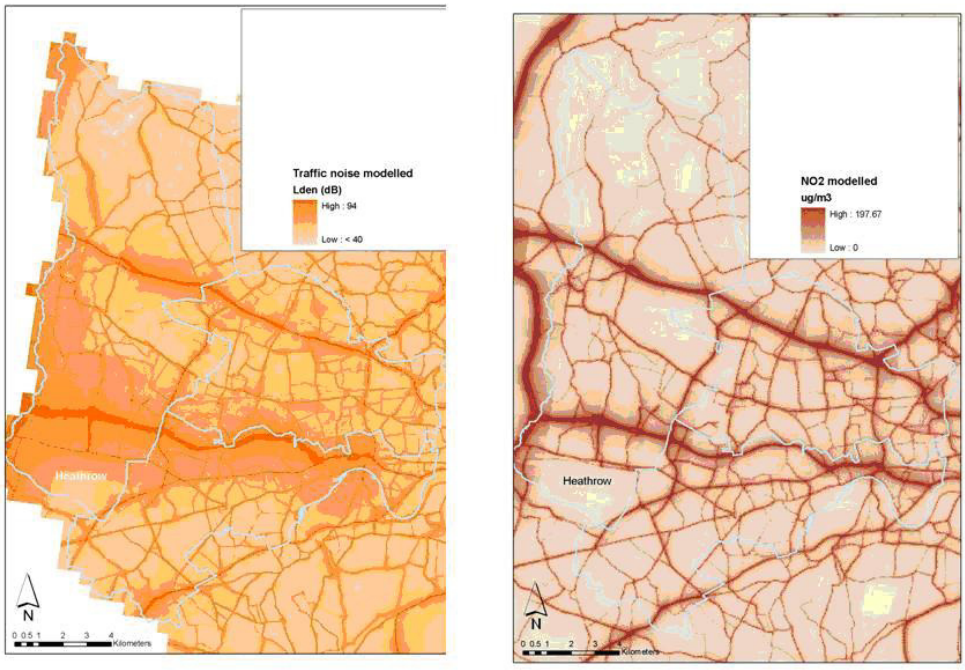
This study aims to examine potential effect modifiers of the impact of noise on health outcomes, with particular focus on air pollution. More detailed exposure assessments to noise and air pollution than used in the original HYENA study will be derived for study participants and for a study area around Heathrow, and these will be used in an individual-level and ecological study to examine whether exposure to ambient air pollution is a confounder or effect modifier of the association between noise and health outcomes.

Data

This project will analyse data collected by the HYENA survey on persons aged 45 to 70 who had lived at least 5 years near a major airport. It is expected that data from at least three countries, UK, Netherlands and Sweden, will be available for analysis. The UK data are on 648 people living near Heathrow airport, the Netherlands data are on 913 people living near Schiphol airport and the Sweden data are on 1025 people living near Arlanda and Bromma airports. Blood pressure measurements were taken on home visits and, at the same time, data on health, socio-economic status and life-style factors were collected via the questionnaire.

For the ecological level analysis, use will be made of data held by the Small Area Health Statistics Unit (SAHSU) on mortality (cardiovascular and respiratory diseases) and hospital admissions (hypertensive disease and asthma).

Figure 8.14.1 Noise and air pollution models (from road traffic) for Ealing, Hounslow and Hillingdon (east of Heathrow)



Source: Small Area Health Statistics Unit, Imperial College London. The air pollution map was produced using the ADMS-Urban model and the noise map was produced using road traffic noise data ('London Road Traffic Noise Map') obtained from Defra

Methods

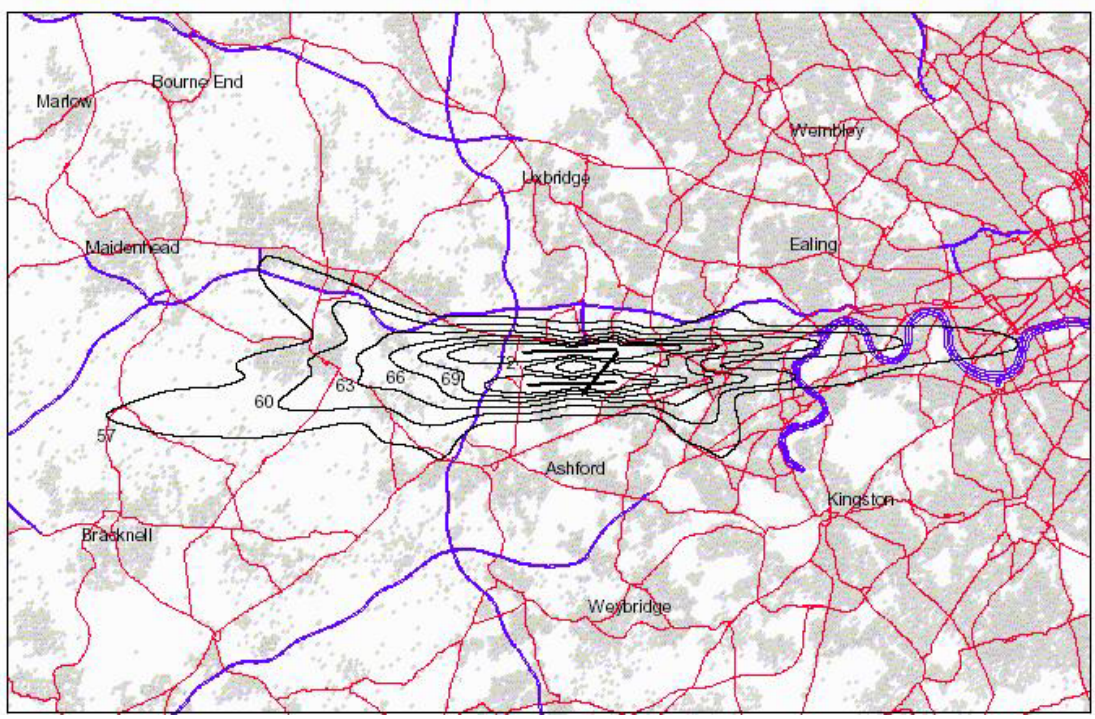
Individual exposure profiles will be created for the HYENA subjects using the information gained in the questionnaire on type of residence, location of residence, orientation of living rooms and bedrooms toward roads, window opening habits, sound insulation and length of residence. This information will be incorporated with noise maps (for both aircraft noise and road traffic noise) for each airport and its surrounds with the aim of better characterising each individual's exposure to noise. Figure 8.14.2 below shows the aircraft noise contours for Heathrow.

Exposure to air pollution for each individual will be assessed based on dispersion models of NO_2 and PM_{10} for each airport and its surrounding area.

Different methods of analysing the joint effects of noise and air pollution will be assessed: for example, treating air pollution as a covariate in a regression model, or stratifying the subjects according to different exposure levels.

In addition to the individual level analyses, risk of cardiovascular and respiratory disease in the population living in the area around Heathrow will be assessed using routinely collected data held by the SAHSU. To assess the relationship between the exposure indices and the health outcomes, an ecological regression will be performed using modelled levels of noise and air pollution and the data held on mortality and hospital admissions.

Figure 8.14.2 Aircraft noise contours 2002 for Heathrow (average mode with Concorde movements at 1999 values 16 hr Leq on population map)



Source: Department for Transport (Noise Exposure Contours for Heathrow 2002)

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8.15 Arrhythmogenic and ischemic effects of particulate air pollution on patients with cardiac disease: Preliminary analysis of ECG parameters

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Background and objectives

Cardiovascular disease is a leading cause of morbidity and mortality in the UK and around the world. Air pollution has been identified as a risk factor. It is estimated that worldwide air pollution accounts for 0.5% disability-adjusted life years and human health costs.

The mechanisms by which the cardiovascular system is affected are not well understood. Two major hypotheses were considered by the Committee on the Medical Effects of Air Pollution (COMEAP, 2006). First, particulate matter (PM) exposure induces changes in inflammatory and thrombotic activity and alters endothelial function. Second, inhaled particles may stimulate receptors in the airway that trigger an autonomic-mediated reflex resulting in changes in the rhythm of the heart.

Heart rate variability (HRV) refers to changes in the time interval between heartbeats. Measurement of HRV by electrocardiogram (ECG) is a non-invasive technique that can be used to quantify cardiac autonomic control. HRV thus lends itself to the exploration of the impact of the autonomic nervous system on the heart. Reduced HRV is indicative of impaired autonomic control. Small but significant decreases in HRV have been observed following exposure to PM_{10} (Pope *et al.,* 1999) and $PM_{2.5}$ (Pope *et al.,* 2004) when PM exposure was assessed using fixed site monitor data. Few studies have used personal monitoring to assess exposure (Magari *et al.,* 2001; Chan, 2004).

Several studies have noted a more pronounced effect on HRV in susceptible subgroups, including the elderly (Chan, 2004) and individuals with cardiovascular disease. These studies provide evidence of a fairly consistent effect of PM on HRV. Although there is now substantial evidence linking air pollution to cardiac changes, especially HRV, there remain uncertainties about the clinical significance and groups most susceptible to exposure to air pollution.

Despite the compelling body of evidence linking PM to changes in HRV, few studies have looked at other potential markers of cardiac changes, such as repolarisation and ischemia parameters. These parameters are also measurable via ECG. One parameter of specific interest is ventricular repolarisation (QT interval). Abnormal ventricular repolarisation (>400 ms) is implicated in sudden lethal arrhythmia (ventricular fibrillation), which is mostly brought on by ischemic disease but sometimes occurs where there is no discernible heart pathology (idiopathic ventricular fibrillation); both potentially exacerbated by air pollution. It is well known that many drugs affect ventricular repolarisation via an action on ion channels of the cardiac cells (Tanaka & Hashimoto, 2007). If air pollution can also be shown to affect repolarisation this could demonstrate a novel mechanism for a cardiac effect and one which might be determined by the chemical constituents of air pollution. To date only a few studies have looked at QT interval as a marker of air pollution impact on the heart, revealing an increase in QT interval in response to organic carbon (Henneberger *et al.,*2005) and traffic related particles (Yue *et al.,*2007) but not in the response to ultra fine particles (Frampton, 2004). The mechanisms linking particulate air pollution and myocardial ischemia are presently unknown. ST-segment depression, an ECG manifestation of ischemia, may be associated with increased exposure to particles.

To explore the hypothesis that patients with cardiac conditions will experience more pronounced cardiac changes, or experience changes at lower levels of exposure than healthy individuals, we will conduct a study to:

- explore the associations between real time changes in particulate matter exposure and arrythmogenic and ischemic effects
- explore the impacts of differing exposure sources on these cardiac changes
- compare the susceptibilities of different cardiac patients
- evaluate the validity of using fixed site pollution monitor data to assign individual level exposure measure.

Study description

The proposed research utilises a case-crossover design to investigate the associations between real time changes in air pollution levels and concurrent changes in relevant ECG parameters, including arrhythmogenic and ischemic effects and changes in HRV, alongside information on physical activity and location. Study participants will be identified from an outpatient cardiology clinic with both patients and healthy volunteers being approached. The intention is to assess the associations between air pollution and ECG parameters under realistic exposure and physiological conditions so it is hoped that participants will follow their normal daily activities.

A better understanding of the impact of exposure on groups with differing susceptibility would be valuable, and as a result our study participants will include the following groups.

- Patients susceptible to arrhythmia
- Patients susceptible to ischemia
- Healthy volunteers without symptoms of heart disease

Informed by previous studies, we aim to recruit up to 30 participants in each group. Our subjects will be monitored for 24 hours, a far greater sampling period than has been previously used. This will allow us to investigate the temporal variability in air pollution/cardiac relationship.

Data will be collected using an ambulatory ECG device, a personal air monitor, an activity monitor and GPS technology that will be worn by study participants for a 24-hour period. In addition to collecting these real time data we will also collect information on each participant's medical history, current medication, lifestyle and household characteristic. During the sampling period we will ask the participants to keep a time–activity diary to provide a context to the GPS and activity monitor data, and to fill out a questionnaire highlighting activities such as exposure to tobacco smoke, alcohol and coffee consumption that may impact on the cardiac system.

As an indication of arrythmogenicity, we will measure QT prolongation (Moss, 1993), ventricular repolarisation heterogeneity (Zabel *et al.,* 2002) and atrial and ventricular ectopy (Frolkis *et al.,* 2003). To assess ischemia we will measure ST elevation/depression (Jernberg *et al.,* 1999), and to assess impacts on the autonomic nervous system we will measure HRV, which will enable comparison with other studies that have assessed this end point.

Preliminary study

We have undertaken an initial study to determine the suitability of our cardiac monitoring equipment for 24-hour measurements and to assess a subset of cardiac ECG parameters that will be used in our main study. Here we present details of this preliminary study.

Method for cardiac monitoring

24-hour electrocardiographic recordings were obtained using a 12-lead Holter monitor (H12+ Mortara Instruments, Italy) from 10 subjects (6 male, 4 female) undergoing normal daily activities. No air quality monitoring was undertaken. ECGs were analysed in the time domain by the ECG analysis programme H Scribe (Mortara Instruments, Italy). Heart rate (HR) and HRV (the square root of the mean of the squared differences between adjacent normal RR intervals, r-MSSD) were calculated. The mean values of the minimum (min), maximum (max) and mean HR and r-MSSD were calculated for each 60-minute segment throughout the entire recording. Within-subject variability was calculated as the standard deviation of the 60-minute mean values. For each subject, we calculated the mean of the 24-hour values and took the standard deviation of the mean to determine between-subject variability.

Preliminary results from EGG data

Table 8.15.1 provides summary statistics for the subject's age and ECG parameters. None of the subjects suffered from a history of heart disease or was on cardiovascular medication. The preliminary recordings show acceptable quality in the recording, and subjects were able to tolerate wearing the ECG device for at least 24 hours without problems except minor skin irritation at electrode sites. Table 8.15.2 shows the results for the within- and between-subject variability.

Table 8.15.1 Mean, minimum, maximum heart rate and heart rate variability (24-hour period)

Total population	
Subjects *n*	10
Age *y*	
Mean	36.5 ± 10.1
Range	21–61
HR, min bpm	44.4 ± 7.9
HR, mean bpm	75.7 ± 7.4
HR, max bpm	180.3 ± 28.1
r-MSSD, mean, ms	69.7 ± 12.5

HR: Heart rate; r-MSSD: root mean square of successive differences in normal RR intervals; bpm: beats per minute; ms: milliseconds
All values are mean (± SD)

Table 8.15.2 Within- and between-subject variability

Outcome	**Within subject**	**Between subject**
Variable		
HR (min)	7.4 ± 2.3	SD 6.8
HR (mean)	11.5 ± 2.8	SD 7.3
HR (max)	25.6 ± 10.2	SD 11.6
r-MSSD	32.1 ± 9.9	SD 12.3

Conclusion and discussion

Our preliminary study shows that our ECG device is practical for 24-hour monitoring and provides acceptable quality of recording for analysis of ECG parameters which will be used in the main study. The measured values are consistent with those reported in the literature for healthy subjects. Our future study will assess cardiac changes that have not been previously been explored in relation to particulate matter exposure, and we will compare cardiac changes in groups of patients with different susceptibilities.

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8.16 Size distribution of fungal and bacterial aerosol in domestic environment

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Introduction

Bioaerosols are ubiquitous in all environments, but the interest in their size and composition in indoor environments has increased significantly in recent years as their association with occupational and environmental health has been acknowledged. There is a reasonable amount of evidence that exposure to biological agents in the indoor environment can have adverse health effects. Recently, a report on indoor air quality and dampness and mould by the WHO (2007) concluded that sufficient epidemiological evidence is available that inhabitants of damp or mouldy buildings, both homes and public buildings, are at increased risk of experiencing respiratory symptoms, respiratory infections and exacerbations of asthma and there is clinical evidence that exposures to moulds and other dampness-related microbial agents increase the risk of rare conditions, such as hypersensitivity, pneumonitis/allergic alveolitis, chronic rhinosinusitis and allergic fungal sinusitis. However, the studies on levels of bioaerosols in various residential settings are rare and sporadic. Most of these studies reported only the total counts of biological agents. But the knowledge of size distribution of bioaerosol in indoor settings is of great importance with regard to their potential health effects and to understand their fate indoors. Therefore, the present investigation was undertaken to assess the size distribution of bioaerosols in the residential houses of the south east of England.

Methods

Twelve single-family residences were sampled in Colchester during the summer of 2007. The houses were of different ages and the samples were taken by using an Anderson six-stage viable particle sampler, loaded with malt extract agar and nutrient agar. The nutrient agar was used for the total bacterial counts while isolation, cultivation and enumeration of fungi were carried out by malt extract agar. The sampling interval was of 5 minutes, and after collection the agar plates were incubated at 25 ºC for 48 hours. The malt extract agar plates were incubated for an additional 24 hours if sporulation did not occur. Data on humidity, temperature, water damage, visible mold, age of home, number of occupants, respiratory illness and pets were recorded. Colony forming units (CFU/m^3) were enumerated for each stage and total counts for all the stages were made.

Results

The concentration of bacterial aerosols was in range of 1102–9780 CFU/m^3 with a mean of 3677 CFU/m^3. The mean CFU/m^3 for fungal aerosols was 1424 CFU/m^3 and varied between 240–3236 CFU/m^3. With regard to total CFU/m^3, the mean concentration was 5102 with a range of 1398–1469 CFU/m^3 (Table 8.16.1). Temperature and humidity were between 20–24 ºC and 36–63%, with a mean of 22 ºC and 49%, respectively.

Table 8.16.1 Summary of bioaerosol concentration, temperature and humidity in 12 single-family residences

	Average	Range
Bacterial CFU/m^3	3677.00	1102–9780
Fungal CFU/m^3	1424	240–3236
Total CFU/m^3	5102	1398–11469
Temperature (ºC)	22	20–24
Relative humidity (%)	49	36–63

On the topic of the percentage of bioaersols in each size range, the majority of bacterial aerosols were recovered from Stage 1 (7 µm & above), Stage 2 (4.7–7 µm), Stage 3 (3.3–4.7 µm), and Stage 4 (2.1–3.3 µm). However in old (>30 years) houses most of them were concentrated in Stage 5 (1.1–2.1µm) and Stage 6 (0.65–1.1 µm).

On the other hand, in most of the houses more than 75% of the fungal colonies were found in stages 2, 3 and 4 (Figure 8.16.1, 8.16.2 & 8.16.3). However, at some residences a reasonable number of colonies were on Stage 5 as well.

Figure 8.16.1 Concentration of viable bioaerosols in various size fractions

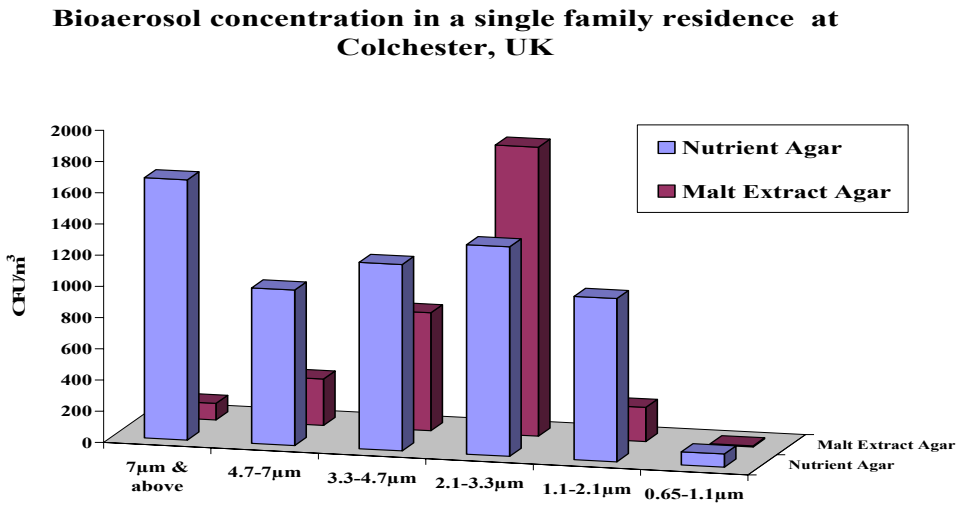


Figure 8.16.2 Fungal CFU/m^3 in different size fractions in a living room

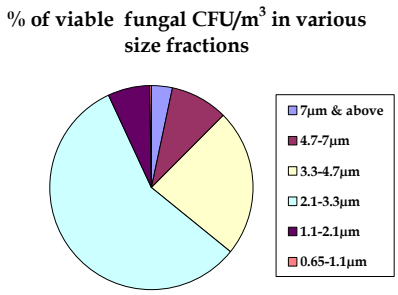
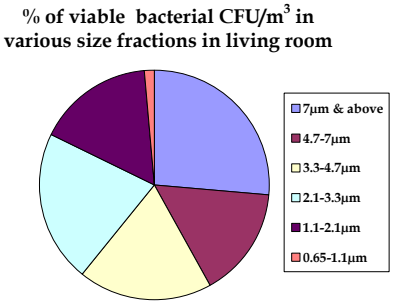


Figure 8.16.3 Bacterial CFU/m^3 in different size fractions in a living room



Conclusions

The concentration of bioaerosols varied over a wide range, and in some cases a considerable presence of biological agent was recorded. Housing characteristics, dampness and structural failures were found as significant variables in indoor concentration of bioaerosols.

This was a small-scale study and hence may not provide a true reflection of a wide range of housing characteristics and geographical locations. Further work is required to establish the levels of bacteria and fungi in the air of domestic dwellings.

References

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9 Closing Remarks

At the end of the meeting Professor Maynard invited Dr Rombout to give a closing address.

Dr Rombout noted the attention given to research in the field of air pollution within the UK and the active government support that it receives. He also stressed the great value of regular workshops at the national level that allowed open exchanges between researchers from a wide range of disciplines, and welcomed the breadth of expertise present at this workshop, ranging as it did from students and postgraduate researchers to internationally recognised experts in the field. Considering the workshop in its entirety, it was apparent that a number of important aspects had been discussed, including:

- the importance of particle surface interactions in elicitation of toxic effects
- the extent of gaps in current understanding of, and apparent importance of, indoor air chemistry
- the need to place unusual regional events (such as Saharan dust storm episodes) in perspective compared with local area pollution episodes
- the need to reconsider the contribution to ill-health made by coarse particulate pollution, rather than focus on $PM_{2.5}$ or smaller fractions
- the need for developed countries to assist the developing nations to develop and enact appropriate strategies to address their pollution issues
- the need to investigate the role played by road dusts other than vehicular pollution on health effects, and finally
- the need for both regulators and scientists to consider the public health and economic implications of air pollution effects, not just in terms of mortality, but also its impact on morbidity and quality of life.

Annex I Workshop Participants

Name	Affiliation
Mohamed Abdallah	University of Birmingham
Emilia Zainal Abidin	University of Aberdeen
David Aldred	Cranfield University
Morris Anglin	Newcastle University
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Nicola Carslaw	University of York
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Jennifer Desborough	University of Birmingham
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Stephen Holgate	University of Southampton
Fintan Hurley	Institute of Occupational Medicine
Catalina Ibarra	University of Birmingham
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Lata Koshy	Cardiff University
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Bob Maynard	Health Protection Agency
John McAughey	British American Tobacco

Name	Affiliation
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Zaheer Ahmad Nasar	University of Essex
Jane Newbold	University of Hertfordshire
Louise Newport	Department of Health
Jack Pease	Air Quality Bulletin
Annette Peters	Helmholtz Zentrum Munich
Heather Price	Cardiff University
Caitríona Ní Riain	Max Fordham LLP
Peter Rombout	RIVM Netherlands
Michael Routledge	University of Leeds
Patrick Saunders	Health Protection Agency
Sean Semple	University of Aberdeen
Rebecca Slack	Institute of Environment & Health, Cranfield University
Terry Tetley	Imperial College London
Andrew Thorley	Imperial College London
Anya Tremper	King's College London
Marcella Ucci	University College London
Max Wallis	Friends of the Earth Cymru
Dafydd Walters	St George's Hospital Medical School
Heather Walton	Health Protection Agency
Giles Watson	Health Protection Agency
Ursula Wells	Department of Health
Andrew Whitcombe	Department of Health
Anna Wlodarczyk	Cardiff University

Annex II Workshop Programme[1]

ELEVENTH ANNUAL UK REVIEW MEETING ON OUTDOOR AND INDOOR AIR POLLUTION RESEARCH

At CMDC, Cranfield University, 15–16 April 2008

Tuesday 15 April

09.00–10.00 **Registration**

10.00–10.10 **Welcome and introduction** (Paul Harrison & Bob Maynard)

10.10–10.35 **Invited keynote presentation – Michaela Kendall**

10.35–11.15 **Atmospheric Chemistry, Exposure Measurement and Modelling** (Chair: Stuart Harrad)

1 Model development and validation of personal exposure to VOC concentrations (MATCH project) – Juana Maria Delgado Saborit

2 Changes in second-hand smoke concentrations in bars following smoke-free legislation in Scotland and England – Sean Semple[2]

11.45–13.00 **Atmospheric Chemistry, Exposure Measurement and Modelling (cont'd)**

3 Pollution infiltration and ventilation design in the urban context – Caitríona Ní Riain

4 An improved model for indoor air chemistry – Nicola Carslaw

5 An overview of indoor contamination with persistent organic pollutants – Stuart Harrad

General Discussion

14.00–14.40 **HPA Annual Air Pollution Research Lecture – Bert Brunekreef** (Chair: Robert Maynard)

14.40–15.20 **Particles and Health** (Chair: Terry Tetley)

1 Alteration of fibrin clot properties by ultrafine particulate matter – Sofian Metassan

2 Relation between respiratory symptoms and lung function in rural and urban Nepalese adults– Om Kurmi[3]

[1] Please note that the titles of presentations given in the programme are those provided by the authors prior to the meeting and may not exactly match those used by the authors at the workshop or in this report.

[2] Jon Ayres gave presentation in place of Sean Semple

[3] Jon Ayres gave presentation in place of Om Kurmi

15.50–17.00 **Particles and Health** (Chair: Terry Tetley)

3 Science and communication: does ultrafine particulate matter from waste incineration affect infant mortality – Mark Broomfield

4 Particulate air pollution and risk of stillbirth: The UK PAMPER study – Svetlana Glinianaia

General discussion

17.00–17.30 **Poster 'quick fire' session** (Chair: Paul Harrison)

17.30–18.00 **Poster viewing**

Wednesday 16 April

09.30–10.00 **Invited keynote presentation – Klea Katsouyanni**

10.00–10.40 **Epidemiology, Public Health and Policy Development** (Chair: Klea Katsouyanni)

1 Links between urban ambient particulate matter and health: Time series analysis of particle metrics – Richard Atkinson

2 Quantification for health impact assessments: Linking deaths, lives and values – Brian Miller

11.10–13.00 **Epidemiology, Public Health and Policy Development (cont'd)**

3 Modelling solvent dispersion, chemistry and impacts to inform policy development on VOC control – Mike Holland

4 An evaluation of the impact of the congestion charging scheme on pollution concentrations in London – Richard Atkinson

5 The public health implications of burning waste derived fuels in cement kilns: A*ssessing the evidence and developing policy – Patrick Saunders*

General discussion

14.00–14.40 **Parallel discussion sessions**

1 Particle Toxicology (Facilitator: Annette Peters)

2 Distributed Lags (Facilitator: Ross Anderson)

14.40–15.00 **Feedback on parallel discussion sessions to plenary**

(Chair: Bob Maynard, Heather Walton)

15.30–16.15 **Final discussion** (Chair: Robert Maynard)

16.15–16.30 **Closing address by Peter Rombout** (Chair: Robert Maynard)

16.30 **Close of meeting**



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