MRC Institute for Environment and Health

Joint Research Programmes on Outdoor and Indoor Air Pollution

A Review of Progress
1999

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Compiled and edited by Jean Emeny and Linda Shuker

IEH would like to thank the researchers participating in the DETR/DH/MRC Joint Research Programmes on Outdoor and Indoor Air Pollution, particularly those contributing to the third annual review meeting on which this report is based. Special thanks are extended to the independent experts who participated in the review meeting and helped to assess the contributions the Joint Research Programmes have made to a better understanding of the impact of air pollution on human health.

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Executive summary

Early in 1994, the Medical Research Council (MRC) Institute for Environment and Health (IEH) began investigating, with the Department of the Environment (now Department of the Environment Transport and the Regions, DETR) and the Department of Health (DH), what the priority areas should be for research into air pollution and respiratory disease in the UK.

Since then, the DETR, DH and MRC have, with the involvement of IEH, supported Joint Research Programmes on outdoor and indoor air pollution. Many leading UK research teams in the field have participated in these programmes. Three annual review meetings have been held; these have enabled participants in the programme to meet each other, to report on progress and to discuss with the sponsors the future direction and policy implications of research on outdoor and indoor air pollution in the UK.

This report presents a detailed account of the third annual review meeting held in March 1999. This was a milestone event as many of the initial projects had come to or were approaching completion, which created an ideal opportunity to review the status of the programmes and to discuss possible future research directions. This report gives an overview of what the Joint Research Programmes have contributed to overall understanding in the field, taking into account other relevant research in the UK and elsewhere. It also looks to the future, both in terms of the need for further research and the possible policy implications of the results from the programmes to date.

A number of areas for future research of particular relevance to the development of UK policy on outdoor and indoor air pollution have been identified.

Exposure

- The relevance of the 'personal cloud' of airborne particles and its impact on total personal exposure
- Exposure modelling and its application (e.g. in epidemiology)
- Exposure measurements for source apportionment
- Clarification of dose–response curves, especially important for cost–benefit analysis and policy implications
- Investigating which substances in or on fine particles are important
- Identification of 'standard particles', the use of which could facilitate comparison between experimental studies

Health effects

- Further studies on the degree of life shortening by air pollution episodes
- The relative importance of air pollution and social disadvantage — is deprivation actually the most potent predictor of hospital admissions?
- The importance of diet as a modifier of response to air pollution
- Investigations of natural experiments (e.g. new road schemes, factory closures)
- Studies of long-term impacts of air pollution

Susceptible groups

- Identifying which sensitivities are important and who are the sensitive individuals

- Investigating marked idiosyncratic effects seen in some individuals in volunteer studies — Are important differences missed by 'measuring the mean'?

Public health impact and policy

- Presentations of air quality information — Smell, visual impact and dust may concern people just as much as the prospect of possible health impacts
- Determining what should be regulated (especially true for particles)
- Identifying the appropriate policy levers to ensure improvements in public health

A collaborative approach

- Consider establishing a multidisciplinary collaborative project for the next phase of research into air pollution and health effects in the UK, to facilitate a more efficient, less fragmented and less competitive approach

1 Introduction

The impact of air pollution on human health, in particular cardiorespiratory disease, continues to be an area of scientific interest and public concern. Over the years, with increasing controls on sources of air pollution but also increases in some sources, notably vehicular traffic, the focus of concern has changed. Large numbers of acute cardiorespiratory deaths like those seen during short-term pollution episodes in the 1950s no longer occur. However, recent data do suggest the possibility of a link between episodes of air pollution and an accelerated onset of cardiorespiratory mortality and morbidity. It is also becoming increasingly recognised that the indoor environment may play a particularly important role in the exposure of an individual to air pollutants, especially as the vast majority of people's time is spent inside buildings. Thus indoor air pollution is becoming an increasingly important area of study.

In 1994, the Institute for Environment and Health (IEH) instigated and hosted a workshop to establish the priority areas for UK research in air pollution and respiratory disease. The workshop was sponsored by the Department of the Environment (now Department of the Environment, Transport and the Regions, DETR), and was held in collaboration with the Department of Health (DH). Participants in the workshop included the chairmen of seven Governmental advisory groups, in existence at the time, that were concerned with the health effects of air pollution: the Advisory Group on the Medical Aspects of Air Pollution Episodes (MAAPE), the Committee on the Medical Effects of Air Pollutants (COMEAP), the Expert Panel on Air Quality Standards (EPAQS), the COMEAP subgroup on asthma and air pollution, the COMEAP subgroup on particles, the Quality of Urban Air Review Group (QUARG) and the Medical Research Council (MRC) Committee on the Toxic Effects of Environment and Workplace (CTHEW) Aetiology of Asthma Working Group.

The tasks of the 1994 workshop were to:

- consider all existing recommendations and proposals for research on links between air pollution and health, excluding cancer, made by expert advisory groups in the field;
- identify gaps in current knowledge about the links between exposure to environmental chemicals and respiratory ill-health and make additional proposals for research to fill these gaps; and
- reach scientific consensus on the research proposals, set research priorities and develop them into a definitive and workable programme.

Ten priority questions were identified, covering acute or proximate effects of exposure to air pollutants (whether from short-term or long-term exposures), long-term consequences of exposure to indoor and outdoor air pollutants, and methodological issues, including monitoring, characterisation of pollutants, exposures and measures of adverse health effects. A review of the workshop and the priority questions was published by IEH as *IEH Report on Air Pollution: UK Research Priorities* (Report R2; see Annex I).

The ten questions were used as the basis for a joint DETR/DH/MRC call for research proposals on 'Air Pollution and Respiratory Disease' in October 1994, which resulted in submission of over 170 outline proposals. The programme was finalised in October 1996. A total of 25 projects were funded at a cost of £3.5 million over 3 years. Several of the projects funded

by this programme are now complete. Staff at IEH have been managing all the joint DETR/DH funded projects under this programme.

A first review meeting was held at the University of Leicester on 10–11 April 1997. Members of all but one of the 25 project teams attended this meeting, together with a number of sponsor representatives and staff from IEH. All teams present made a short oral presentation in sessions covering exposure, mechanisms, volunteer studies, epidemiological studies and public impact. Some of the teams also made poster presentations.

A second call for research was issued in August 1997. This was for a joint DETR and DH research programme on 'Health Effects of Exposure to Air Pollutants and Damp in the Home' (see Annex II) initiated as result of identifying gaps in the research funded by the first programme. The number of applicants to this programme was 108; 14 projects were funded, at an additional cost of £1.3 million*.

A second review meeting, again hosted by IEH, took place on 28–30 April 1998 at Leicester University, with a similar format to the first meeting. Progress on projects in both Joint Research Programmes was presented and discussed and a number of projects funded by DETR outside the joint programmes in the area of air pollution and indoor air quality were also represented. Some of the project teams also presented posters describing other projects of relevance to the Joint Research Programmes. In all, 36 oral and 15 poster presentations were made.

At the third annual review meeting, held on 29–31 March 1999, 41 projects were presented, either orally or as posters, and discussed by 98 participants over three days. Importantly, this meeting also incorporated discussion sessions with invited expert panellists to discuss the key issues and to assess the contributions of the Joint Research Programmes to date.

The proceedings of the third annual meeting are presented in this report. Abstracts of all presentations made at the meeting are included in Sections 2–6, which cover exposure and modelling, mechanisms, volunteer studies, epidemiology and public health impact. Where projects had already been completed by the second annual review meeting, or where there had been no substantial progress to report since the second meeting, the abstracts included are those presented at the second meeting. A few projects not funded directly by the Joint Research Programmes but closely related to them were presented at the annual review meetings and some of these are also included. Section 7 provides a summary of the discussions about the Joint Research Programmes that took place during the third annual meeting; this focuses on:

- the contribution the Joint Research Programmes have made to ongoing research on air pollution and respiratory disease both within the UK and internationally;
- gaps in knowledge and the direction of future research; and
- possible policy implications of research to date, and proposals for future research, in particular to advance UK policy.

This report provides a complete review of the Joint Research Programmes as of March 1999. A summary description of all projects funded by the Joint Research Programmes is presented in Annex III. The titles of the abstracts presented at the first and second annual review meetings are listed in Annexes IV and V.

The third annual review meeting was attended by researchers, Government representatives, independent experts from the UK and elsewhere in Europe, and staff from IEH. A full list of attendees is provided in Annex VI. A bibliography of the published literature arising from the programmes is given in Annex VII.

Subsequent to the third annual review meeting in March, at the request of DH, a meeting was held at IEH on 22 October 1999 to review further the progress and achievements of the Joint Research Programmes, focusing particularly on the first programme, which had reached the end of its funding, with most of the projects completed or nearing completion. The October meeting reviewed both the policy and scientific impacts of the programme. A summary of the conclusions of the review

* Subsequent to the establishment of the two Joint Research Programmes, one of the projects funded in the first round has been extended and carried forward with an MRC Strategic Grant

meeting, highlighting achievements, potential developments and directions for future research, is presented in Annex VIII. Participants at the October review meeting (listed also in Annex VIII) included representatives from the Joint Research Programmes' funding departments and independent experts on air pollution.

2 Exposure and modelling

2.1 Indoor exposure to air pollutants

2.1.1 Particulate matter (PM_{10}) and benzene in the indoor environment

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November 1996–November 1999; January 1998–July 2000; First and Second Joint Research Programmes

Introduction

Indoor air pollutants are derived from a wide range of sources, including the occupants and their activities (e.g. smoking, cooking, vacuuming), heating appliances, furnishings, building materials and penetration of contaminated air from outdoors. As adverse health effects of air pollution depend on the exposure of the individual it is important to determine indoor concentrations of toxic pollutants and to evaluate individual exposure to these pollutants.

Sampling rationale

Sampling was carried out over 5 day periods at 16 domestic homes. This was completed over a period of 18 months, from May 1997 to October 1998. With such a relatively small study it was not possible to represent the typical housing stock in the UK but houses were chosen from the following five groups: flats; terraced houses; semi-detached houses in town; houses in rural locations; and houses located adjacent to busy roads.

In total 36 sampling periods were completed giving a data set that spanned all seasons and house types.

Methodology

The Tapered Element Oscillating Microbalance (TEOM) was the main instrument used for measuring the concentration of particulate matter <10 µm aerodynamic diameter (PM_{10}). This is a direct reading mass monitor and has the advantage of being able to collect data with relatively short averaging times (10 min), allowing elevation due to activities, such as cooking, that might not affect the daily mean by a significant enough amount to be detected. Personal PM_{10} samples were collected using a personal foam thoracic sampler and were analysed gravimetrically.

In addition to these measurements, an Andersen Cascade Impactor and a Micro Orifice Uniform Depositing Impactor (MOUDI) were used to collect samples. The filters from these instruments were analysed for chemical composition.

Benzene concentrations were determined using an active sampling adsorption technique. Air was pumped through Tenax TA sorbent tubes at a rate of 100 ml/min for 1 hour periods. This method was used to collect both static and personal samples. After sampling, the tubes were thermally desorbed by being heated at 250 ºC for 30 min and eluted with helium to be preconcentrated in a cryogenic trap at –30 ºC using a Perkin Elmer ATD 50 automated thermal desorber. After this the trap was rapidly heated to 250 ºC. Desorbed compounds were transferred to a gas chromatograph for subsequent detection using a flame ionisation detector.

Results

PM_{10} levels measured using the TEOM showed fairly constant background levels with clear peaks associated with cooking and smoking. For cooking periods, peaks were directly associated with the activity of cooking and not just the type of cooking system. The largest elevations were seen for cooking involving fat (either oil or butter) on the hob, e.g. stir-fry.

There appeared to be little seasonal variation in the average static PM_{10} measured using the TEOM. When the data were looked at in terms of house type the average mass concentration was approximately 20 $\mu g/m^3$ for all house types except roadside houses. These had an average static mass concentration of around 25 $\mu g/m^3$.

The Andersen and MOUDI data showed that sulphate tended to be the dominant chemical species in the static particulate matter. There is evidence to show that this was derived from penetration of outdoor air into the home.

Personal PM_{10} levels were always higher than their equivalent static levels. This was due to resuspension of particles caused by the volunteer's activities and the volunteer being closer to point sources (e.g. cooker or cigarette) than the static monitoring equipment. A good relationship between the personal and the static data was observed.

Benzene levels tended to be reasonably low (below 3 ppb) for both static and personal measurements. Very little variation was seen with either house type or season. The static and personal levels were very well related and the overall ratio seen was 1.2.

2.1.2 Indoor-generated particles and their toxicity: A preliminary report

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April 1998–March 2000; Second Joint Research Programme

Introduction

Epidemiological studies have indicated that there is an association between a population's exposure to small particles and rates of morbidity and mortality. In particular, increased death rates occur among older people when outdoor particle concentrations rise. However, people spend more than 90% of their time indoors. The question arises, therefore, as to how far total exposures to particles reflect measurements of outdoor air pollution, how far they are influenced by local indoor sources and how relevant are indoor-generated particles, e.g. from cooking, in terms of adverse health effects.

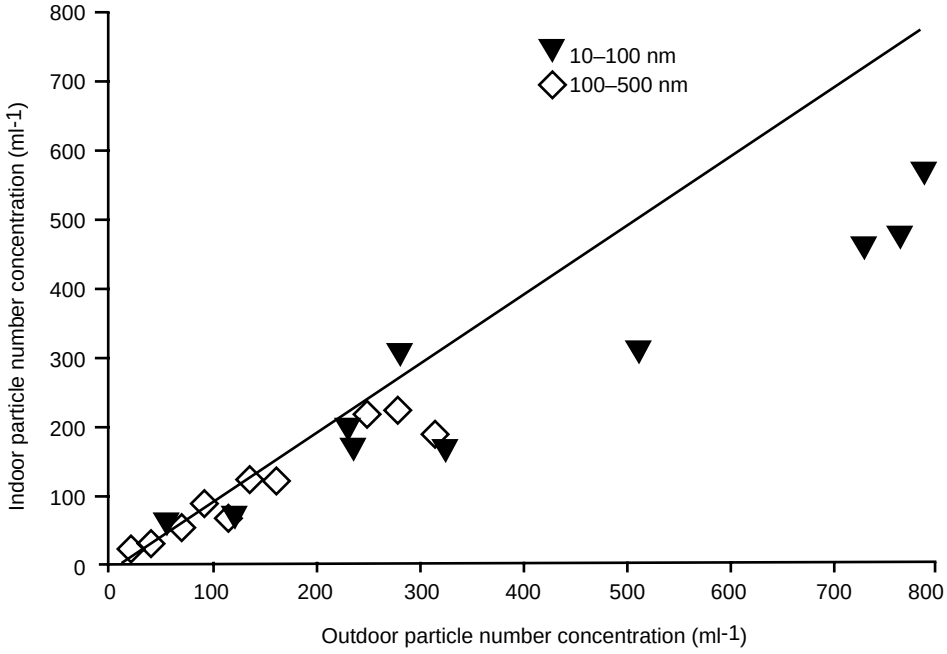
Measurement of particle number and mass concentrations

A TSI 3934 Scanning Mobility Particle Sizer (SMPS) is being used to measure sub-micron particle number concentration and size distribution, and personal PM_{10} and $PM_{2.5}$ sampling heads to measure gravimetric concentration. It is also planned to use a Monitor Labs chemiluminescent NO_x (nitrogen oxides) analyser to measure NO_2 concentration.

Initial work has shown that there are only small losses of nanometre size particles in a 4 mm diameter copper sampling tube used with the TSI monitor, provided the tube is kept straight. Curving the tube in a 180° arc of diameter approximately 1 m produced losses of about 25%, with greater losses for particles smaller than about 50 nm diameter.

Experiments in a room of internal volume 80 m^3 with two large windows and no internal particle sources have shown that the particle number concentrations indoors are consistently lower than outdoors, particularly for smaller particles (Figure 2.1.2a). The indoor particle number concentrations are approximately 30% lower than the corresponding outdoor concentrations.

Figure 2.1.2a Ratio of indoor to outdoor particle number concentration by size category



Preliminary work has shown that the concentration of particles of between 10 and 25 nm in size increases rapidly when a gas cooker is switched on (Figure 2.1.2b). Within about 15 min of starting four burners on a domestic gas cooker the concentration of 10 nm particles had increased by more than 30 times. The concentration of these particles remained elevated in the air of the room for more than 30 min after the gas had been turned off.

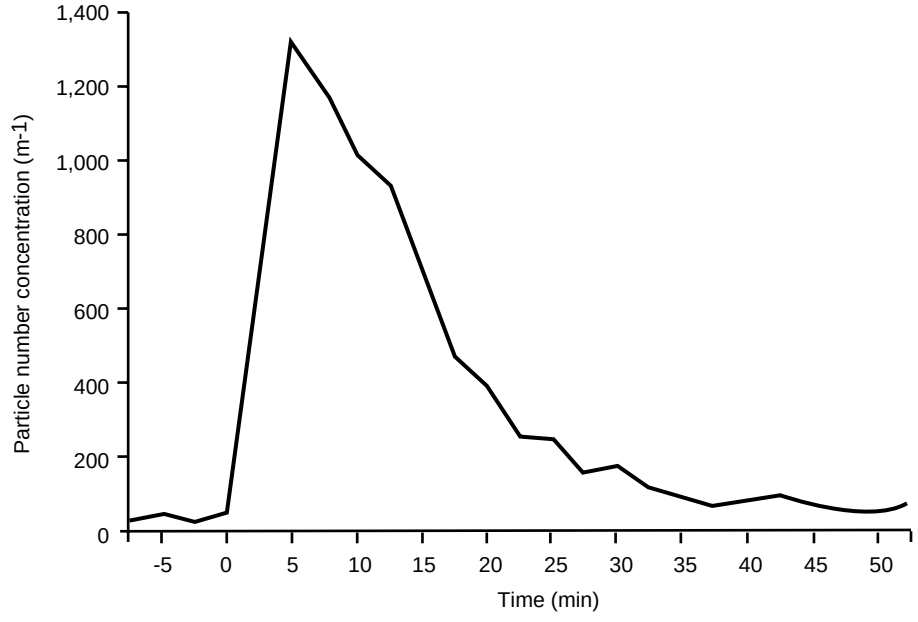
Preliminary work to assess particle toxicity

The free radical activity of outdoor and indoor particles and particles generated during cooking will be investigated by measuring their ability to break supercoiled plasmid DNA and their action on 16 HBE epithelial cells. Preliminary work has concentrated on selection of a suitable air sampling filter. The only filter that does not have significant activity in the biological assays and is suitable in other respects is the Gelman Teflon filter.

Discussion

The concentration of particles found indoors is lower than outdoors, provided there are no indoor sources. This is most likely due to deposition of particles on indoor surfaces rather than the building providing a barrier to particle infiltration. There is substantial emission of nanometre size particles from gas cookers without any cooking taking place. While a cooker is switched on the particle emissions will greatly increase the occupants' exposure to nanometre size particles.

Figure 2.1.2b Particle number concentration generated by a gas cooker



Gas switched on between 0 and 15 min

2.1.3 Size-selective sampling of indoor air exposure to tobacco smoke and allergens

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March 1996–September 1999; First Joint Research Programme

Introduction

There is much evidence that mortality and morbidity are enhanced by episodes of elevated particle concentration, particularly in population groups with existing cardiorespiratory disease (reviewed by COMEAP, 1995a,b, 1998). However, uncertainty in population dosimetry remains and this study is designed to quantify the effects of:

- indoor personal exposure — 85–90% of time is spent indoors; personal exposure may be significantly greater than estimates based on automatic urban network (AUN) PM_{10} data;
- exposure to biological particles — exposure to specific allergens may increase symptoms in atopic populations;
- exposure to significant particle sources — tobacco smoke and penetration from outdoors; and
- the relative importance of each in size-segregated samples.

Study design

Sampling is being conducted in nonsmoking and smoking households, prescreened for cat and house dust mite allergens. During a 1 year period, a series of measurements is being carried out for:

- allergen content (Der p1, Fel d1 and grass) of air in the breathing zone (as proxy for inhaled air) using size-selective personal monitors for 10, 2.5 and 1.0 µm (PIDS, 2 l/min);
- allergen content of ambient indoor air (using size-selective static Andersen cascade impactors in living room and bedroom, 28.3 l/min) at 10, 2.5 and 1.0 µm cut-offs;
- allergen content of dust reservoirs;
- total dust mass and environmental tobacco smoke (ETS) in each of the above samples;
- temperature, humidity, air exchange rate; and
- activity log for rooms with samplers.

In addition, a series of real-time short-term size-selective samples will be taken indoors and out.

Results

A size-selective personal sampler and an Andersen impactor have been modified to produce cut-off points at approximately 1.0, 2.5 and 10 µm, and have subsequently been calibrated. A series of laboratory-based studies has been conducted in an environmental chamber and in the field to evaluate sampler performance.

Sampler performance

The personal samplers were operated over periods of 24, 48 and 72 hours, in parallel with Andersen samplers, in the presence of smoke and allergenic material. The samplers showed good agreement on mass concentration and on the fractional mass recorded within each size band. Good agreement was observed for measured concentrations of tobacco smoke and cat allergen (Fel d1), with good linearity over each incremental day of field sampling. The results for house dust mite allergen (Der p1) were consistent but uncertainties were greater owing to lesser amounts of material being airborne.

No cross-interference was observed in the tobacco smoke and allergen assays, and no problems were encountered recovering the particulate tobacco smoke material from the gels used for allergen capture. The field data indicate that a sampling period of 48 hours is feasible, but 72 hours is optimal with respect to improving analytical certainty in the assays. Amplification assays allow a limit of detection for Fel d1 allergen of 0.01 µg. It should be noted, however, that insufficient mass can be collected on the personal sampler for reproducible measurement (as was predicted before commencement).

Activity monitoring

Pilot studies indicated a degree of resistance to instillation of a TEOM in individual homes for indoor versus outdoor measurements. Thus an alternative system (Condensation Particle Counter) has been modified and deployed, such that real-time particle counts (for episodes), integrated mass, particle size distribution (static Andersen samplers), temperature and humidity are monitored and data logged simultaneously (indoor and outdoor). The full apparatus for indoor measurements is contained in a 1 m^3 wooden box that offers a degree of soundproofing for the pumps and is much more acceptable. The use of particle counting offers a more sensitive real-time measurement of airborne particles, which offers scope for mass estimation for activities noted on questionnaires.

Study homes

A mixture of study homes (smokers and nonsmokers, but all having at least one cat) have been screened and data on home construction, occupancy, cooking habits, smoking habits and respiratory health collected by questionnaire.

Der p1 concentrations averaged 8.5 µg/g (0.1–41 µg/g) across the study homes with Fel d1 levels averaging 580 µg/g (12–2500 µg/g).

A number of surveys have been conducted to optimise compliance with questionnaires and monitoring and operating procedures for all equipment prepared.

References

COMEAP (1995a) *Asthma and Outdoor Air Pollution*, London, HMSO, Committee on the Medical Effects of Air Pollution

COMEAP (1995b) *Non-biological Particles and Health,* London, HMSO, Committee on the Medical Effects of Air Pollution

COMEAP (1998) *Quantification of the Effects of Air Pollution in the United Kingdom,* London, The Stationery Office, Committee on the Medical Effects of Air Pollution

2.1.4 The effect on health of exposure to air pollution and damp in the home — air quality in Solihull, West Midlands

S Marks and G Pool

BG Technology, Gas Research & Technology Centre, Loughborough

January 1998–March 1999; Second Joint Research Programme

Introduction

Personal exposure to a variety of airborne contaminants in the home is acknowledged to present a potential risk to an occupant's health. This risk is determined both by the amount of pollutant present and the length of time for which an individual is exposed.

The use of gas-fired appliances in the home generates two gaseous products of combustion, carbon monoxide (CO) and NO_2, which can affect health if inhaled in sufficient quantity. There have been few studies aimed at measuring the level of these contaminants in UK households and it is desirable to identify the extent of such indoor air pollution.

This project involves measuring CO and NO_2 levels in households and obtaining some evidence of personal exposure levels and health effects.

Methods

The levels of CO and NO_2 are being measured in 80 homes owned by Solihull Council in the West Midlands. The composition of the sample population is representative of four specific subgroups: gas users/non-gas users, smokers/nonsmokers, new/old house (built pre-1965) and asthmatics/non-asthmatics. Participants are being selected from the 12 000 or so houses that the Council owns and with the Council's full cooperation.

With the assistance of the Council, portable monitors are being placed in the homes of participants for a 5 day period, including a weekend, during which time data will be logged at 1 min intervals. The CO, NO_2, and O_2 concentrations will be measured using sensitive electrochemical cells within the monitor along with relative humidity, temperature and personal activity within a room (via a passive infrared detector). The monitor is normally being placed in the room in which the participants spend most of their time.

The survey also involves an assessment of the participants' blood carboxyhaemoglobin (CoHb) levels at the start of the survey period using a Bedfont EC50 ToxCO breath CO monitor.

Initially each participant is interviewed using a detailed questionnaire in which information on household members, house details (e.g. type, age, double glazing, cavity insulation), and details of all gas/non-gas appliances (e.g. age, model, service history) are recorded.

An assessment of the CO levels outside each dwelling is made using simple Draeger tubes to indicate peak levels over each 5 day period. These provide an indication (to within ±15% for this time period) of levels existing outdoors and these can be compared with background levels measured indoors.

Preliminary data analysis

To date 73 homes have been surveyed over a 5 day period, resulting in around 7200 data sets for each house surveyed (Table 2.1.4a). A number of summaries of the data were obtained in order to enable meaningful analysis. The mean average and maximum observed CO and NO_2 levels were derived for each house. In addition, rolling averages were calculated for each minute-by-minute reading for 15 min, 30 min, 60 min and 8 hours, to enable continuous comparison with the World Health Organization (WHO) guideline limits.

Individual variables have been investigated for any association with increased mean pollutant levels of both CO and NO_2. Stepwise multiple regression was used to identify the most important variables and to investigate factors associated with differing rates of build-up and/or dispersal.

Secondary analyses included the investigation of any association between exposure to CO and COHb levels, and any factors associated with increased rates of asthma in the household.

Results

Households were classified as 'gas users' if there was a gas appliance in the same room as the monitor unless the only gas appliance was a boiler with a room-sealed flue. All other households were classified as 'non-gas users'. Analysis was stratified between those houses in which the monitor was located in the kitchen and those in which it was located in a different room (usually the lounge).

Sixty-three houses, of which 50 were classified as gas users, had the monitor located in the kitchen. Preliminary analyses of this sample indicate that gas users tended to have higher mean levels of pollutants than non-gas users. This trend was statistically significant at the 5% level for NO_2, but not for CO. Peak values for both pollutants were significantly higher for gas users than for non-gas users. Ten houses, of which seven were gas users and three were non-gas users, had the monitor located in the lounge or dining room. All the non-gas users had solid fuel appliances. Preliminary analyses indicate that non-gas users tended to have higher levels of NO_2, although there was no difference between the overall levels of CO measured.

Of all the properties surveyed 20% exceeded the WHO guideline limit for exposure to NO_2 at some point in the study (Table 2.1.4b). All of these houses were classified as gas users, and all had gas cookers in the same room as the monitor. Houses in which the guideline limit had been exceeded tended to have an older cooker; this trend is borderline significant.

The two properties that exceeded the guideline limit for 60 min CO were both classified as gas users, each having a monitor within 1 m of the gas cooker. The gas cookers in these properties were both at least 15 years old. One of these properties was also the property for which the 8 hour CO limit was exceeded. This particular property had by far the highest mean CO reading of all the houses over the duration of the study.

The build-up and dispersal rates of NO_2 were investigated to identify any contributory factors. In fact, there was no relationship between the rates of build-up and dispersal of individual peaks. The age of the house and double glazing did not appear to have any effect on either rate. Smaller kitchens appeared to have quicker build-up rates, but there was no effect on the dispersal rate. Visually it appeared as though the houses with smokers might have had slower build-up rates and dispersal rates, although neither of these were significantly different.

Table 2.1.4a Results for the first 73 houses surveyed

Pollutant	Level	Mean	Median	Minimum	Maximum
NO_2	Average	6.12 ppb	4.82 ppb	0.2 ppb	25.3 ppb
	Peak	203 ppb	156 ppb	20 ppb	865 ppb
CO	Average	0.45 ppm	0.32 ppm	0 ppm	2.27 ppm
	Peak	11.5 ppm	6.0 ppm	0 ppm	142 ppm

Mean is the mean of all of the data recorded and peak values are the highest pollutant levels recorded for 1 min or more during the surveyed period

Table 2.1.4b Number of houses that exceeded the WHO guideline limits for exposure

Pollutant	Exposure	WHO guideline	Houses exceeding limit	
			Number	Per cent
NO_2	60 min	105 ppb	15	20.5
CO	15 min	90 ppm	0	0.0
	30 min	50 ppm	0	0.0
	60 min	25 ppm	2	2.7
	8 hours	10 ppm	1	1.4

The most important predictor of COHb, as measured by a breath test, was the smoker status of an individual. There was a very clear association between smoking and an increased level of COHb: 80% of those providing readings higher than 5 ppm CO (0.4% COHb saturation) were smokers. No other factors, including the mean or peak levels of CO recorded in the house, appeared to have had an effect.

The proportion of residents who reported suffering from symptoms of asthma was the same for occupants of gas and non gas homes (17% and 18% respectively). There was no significant association between the use of the gas in the home and the prevalence of asthma symptoms.

Further work

The survey is not yet complete, with 73 out of 80 houses having been surveyed. Data analysis such as data mining (to identify trends) and logistic regression (to determine the interaction between influencing environmental factors such as usage of gas, smoker status, type of house, position) will be carried out at the end of the data collection and any trends identified will be investigated further.

2.2 Personal exposure measurements

2.2.1 EXPOLIS Oxford: Determinants and distribution of personal indoor airborne pollutants in urban populations*

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June 1998–May 2000; Second Joint Research Programme

Introduction

The EXPOLIS Oxford study was designed to provide detailed information on exposure patterns, personal exposure, and indoor and outdoor microenvironmental concentrations of $PM_{2.5}$, volatile organic compounds (VOCs), CO and NO_2. The study takes advantage of the information and experience gathered by two ongoing projects: the EXPOLIS Europe study in six European cities and a study of traffic flow intervention, air quality and health effects in Oxford (EMITS).

The aims of EXPOLIS Oxford are to assess:

- frequency distributions of personal air pollution to $PM_{2.5}$, VOCs, CO and NO_2 in an urban population;
- the time spent by the population in different microenvironments;
- statistical associations between exposure to these different pollutants;
- the determinants of these personal air pollution exposures (housing characteristics, cooker, smoking, indoor and outdoor pollution levels); and
- the contribution of different air pollution sources to personal air pollution exposures.

Methods

The study is being carried out in Oxford in three populations: children, adults and the elderly. From each age group, semi-randomly selected individuals are monitored and keep 15 min activity diaries, for 48 hour periods. Children and elderly wear NO_2 sampling badges. Adults carry equipment sampling $PM_{2.5}$, VOCs, NO_2 and CO. The sampling methodologies have been designed and validated by EXPOLIS Europe, and results are consequently directly comparable.

* We acknowledge the support of the UK Government, Department of Health (DH) and Department of the Environment, Transport and the Regions (DETR; Contract: EPG 1/5/106)

2.2.2 Personal exposure to air pollutants in Hertfordshire, England*

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Introduction

This 12 month study was undertaken on behalf of the Hertfordshire Environmental Forum and the DETR (Chemicals and Biotechnology Division) to investigate the relationship between the levels of air pollutants and the total exposure to air pollutants experienced by individuals as they go about their normal lives in homes, offices, cars and other environments. The study involved monitoring of VOCs, NO_2, formaldehyde and CO and involved 24 employees of Hertfordshire County Council (HCC) and 6 employees of the Building Research Establishment (BRE). Particular emphasis in the study was given to the assessment of exposure of people to pollutants during their normal journey to work.

Methods

The 24 HCC participants were divided into four groups of six. Two groups were monitored in summer and two in winter. The BRE group was studied in both summer and winter. Each participant placed diffusive air samplers in their home, outdoors, at the office, and in a car during a 4 week period. They also wore samplers attached to clothing and placed them at the bedside at night in order to monitor their personal exposure to pollutants. The samplers used were a stainless steel adsorbent tube packed with Tenax TA to measure VOCs, a GMD 570 formaldehyde badge, a Palmes tube for NO_2 and a Drager indicator tube for CO.

One journey to and from work was monitored to measure pollutants in transport using pumped samplers to determine VOCs, including formaldehyde and acetaldehyde. The BRE employees monitored up to five journeys each to and from work in order to examine day-to-day fluctuations in concentrations. A group of 10 HCC employees additionally undertook continuous monitoring of their personal exposure to CO and/or NO_2 over a 24 hour period. All participants kept a diary over a 7 day period to record the time spent in different microenvironments and provided information about their home, workplace and journeys to work.

Results and discussion

The time–activity diaries showed that all but two individuals spent over 80% of their time in buildings. Most of the time indoors was spent in the home and the bedroom was used for 25 to 53% of the total time. The BRE employees recorded the time spent in different environments in different seasons. Time spent outdoors in the summer months (mean 4.8%) was only slightly higher than in the winter months (mean 2.7%).

It was found that exposure to several VOCs, including benzene, is typically higher when travelling by car than using other modes of transport. While the sample size was small, there was a trend in the concentration data: train<bicycle<bus = walking<car. Highest concentrations of petrol-related VOCs occurred in older cars. Cars could also have elevated concentrations of other VOCs derived from other sources such as materials in the passenger and driver cabin. Formaldehyde and acetaldehyde concentrations during travel were higher than reported background concentrations and comparable to those often found indoors. The limited continuous monitoring study of CO and NO_2 found elevated personal concentrations during travel, particularly for CO during commuting by car. One of the two individuals who undertook cooking by gas during the period of continuous monitoring was exposed to NO_2 levels in excess of the 1 hour WHO guideline value.

Using the fixed-site data obtained by diffusive samplers and the time–activity data, it was possible to estimate the proportion of the total personal exposure to pollutants resulting from time spent in different environments. For the study group as a whole the majority of the total personal exposure (80% or more) to all pollutants occurred during time spent indoors. There were differences between individuals and pollutants but indoor environments, and the home in particular, were

* Not funded directly as part of Joint Research Programmes

We thank N Hewitt and L Godfrey at HCC for assistance with administering the study

the most important environments determining the amount of people's exposure to the pollutants monitored.

The fixed-site monitors and time–activity diaries gave results that were within ±50% of the measured personal exposure for the majority of people for all pollutants monitored. The closest agreement was for formaldehyde for which only two individuals differed by more than 50%. This might be because of fluctuations in concentrations of formaldehyde that result from people's activities being lower than for some other pollutants.

For several individuals, exposure to some pollutants during travel was an important fraction of their total exposure. For seven participants the time spent in a car accounted for over 25% of their total exposure to benzene and for the individual with the highest in-car concentration it accounted for 76% of the exposure. This was the oldest car in the study. A similar situation occurred for these participants for exposure to the other VOCs that were monitored and that are contained in petrol vapour and vehicle exhausts. For these individuals therefore, travelling by a different mode of travel or perhaps using a car with lower concentrations of pollutants in the interior could significantly reduce their total personal exposure. However, further work is required that could compare different modes of travel and the use of different car types over comparable journeys to evaluate fully the likely benefits for reduced personal exposure to air pollutants.

It was concluded that exposures of employees of HCC and the BRE to air pollutants are strongly influenced by the air quality in buildings, particularly that in the home. This is a function of the concentration of the pollutant in the air and the time spent in different environments.

2.2.3 Measurement of personal exposure to PM_{10} in the non-workplace environment using passive sampling techniques

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September 1998–August 1999; Second Joint Research Programme

It is recognised that individual exposure to particulates among the general population varies greatly from the levels reported at fixed sites. Efforts to improve understanding of this variation using methods such as microenvironmental monitoring have proved to be problematic.

This pilot study has been designed to investigate the use of a possible tool for measurement of personal exposure. The study is concerned with the application of a *passive* personal sampler (Brown & Wake, 1992), originally developed for use in the workplace, to provide measurements of long-term average exposure. This simple device is small, lightweight and unobtrusive, and requires no power supply or pump and no operator attention during the collection period. Potentially it may be used either as a personal or as a static sampler. The main objective of the study is to determine the relationship(s) between the mass collected by the passive sampler, used as a static sampler, and the concentration measured by pumped PM_{10} samplers for a range of microenvironments appropriate to non-workplace exposure. A supplementary objective is to quantify the variability associated with measurements made with the new sampler.

The study design specifies side-by-side measurements with the passive sampler and a pumped 'conventional' PM_{10} sampler in three microenvironments: (i) outdoor ambient, (ii) indoor domestic kitchen and (iii) indoor domestic living, with two examples of each. In each microenvironment, a minimum of 10 pairs of measurements is being made with exposure times ranging from 1 to 2 weeks. Data obtained will be analysed to quantify the relationships between the two measures and to determine whether the difference (or ratio) between passive and pumped samples is constant across environments.

The main outcomes from this study will be relationship(s) that may be used to derive estimates of PM_{10} concentrations from masses collected by the passive sampler in the specified microenvironments, information about the PM_{10} concentrations prevailing in these microenvironments and information on the variability of the passive sampler.

Successful completion of this study will provide a simple instrument with widespread potential for the measurement of personal exposure of individuals both directly and indirectly over long time periods.

Reference

Brown RC & Wake D (1992) A passive sampler for airborne dust using an electret. *J Aerosol Sci*, *23* (*Suppl*), S623–S626

2.2.4 Personal exposure measurements of the general public to atmospheric particles*

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[2] AEA Technology, Harwell, Didcot, Oxfordshire
[3] Environmental Pollution Unit, Birmingham City Council

December 1994–March 1996; First Joint Research Programme

Objectives

The main objective of this project was to assess the personal exposure of the general public to the thoracic (PM_{10}) fraction of atmospheric particles. Using this information an assessment was made of the validity of estimates of exposure of the general public obtained from particle concentration data provided by the fixed AUN sites.

Methods

The study involved measuring the personal exposures of 30 members of the general public. The criterion for the selection of the subjects (all volunteers) was that they spend some, or all, of their time in the vicinity of one of the two AUN sites within the city of Birmingham. The subjects wore prototype thoracic aerosol samplers for five 2 day periods of sampling during each of three surveys carried out in August/September 1995, November/December 1995, and February/March 1996. The lapel-mounted sampling head consisted of an inhalable aerosol entry and porous-foam particle-size selector to collect the thoracic fraction. Suction at 2 l/min was provided by a small, lightweight sampling pump carried in a small case around the subject's chest. Details of the subject's lifestyle and living conditions were obtained via a questionnaire, and daily activities logged in a diary. Mean PM_{10} levels for the period of each personal sample were obtained from the two AUN TEOM particle monitors for comparison of exposure measurements.

Results and conclusion

The results showed that there was wide variability in individual exposure estimates of members of the general public in Birmingham to the thoracic fraction of ambient aerosol. Despite there being over 300 individual personal exposure measurements obtained with personal samplers, there was very little statistically significant correlation between personal exposure levels and those obtained from PM_{10} levels at the two AUN sites for the same sampling periods. There was some evidence that where a person lived and worked affected this correlation, as did the smoking status of the subject.

An improved relationship was found when the data were considered longitudinally, rather than cross-sectionally.

Reference

Mark D, Upton SL, Lyons CP, Appleby R, Dyment EJ, Griffiths WD & Fox AA (1997) Personal exposure measurements of the general public to atmospheric particles. *Ann Occup Expo, 41 (suppl 1),* 700–706

* Completed by April 1998, abstract presented at second annual review meeting

2.2.5 Carbon monoxide and nitric oxide exposure in vulnerable elderly people

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March 1998–August 1999; Second Joint Research Programme

Introduction

This project investigates the hypothesis that chronic exposure to low levels of CO and nitric oxide (NO) influences the health of the individual. As a result, we might expect exposure to these gases to be greater in individuals who are dependent or unwell. There may also be specific relationships between exposure and particular diseases. However, these relationships cannot be investigated because the variability of exposure to these gases in elderly people is unknown. Therefore, we aim to compare the level of exposure to CO and NO between the vulnerable (dependent or unwell) elderly and the well elderly.

Methods

Hospital study

Elderly people with acute medical problems who are admitted to the geriatric receiving unit represent the vulnerable elderly people. The control group is composed of people admitted for elective orthopaedic surgery but who are not housebound and who are otherwise well. These represent the well elderly people. In hospital, blood is sampled from both the vulnerable elderly and the well elderly on admission. At the same time, a smoking history is obtained from each subject. Each blood sample is analysed using a Radiometer OSM3 co-oximeter for COHb and methaemoglobin (MetHb) levels. Mann-Whitney U tests are used to compare groups.

Community study

In the community, CO and NO data-loggers (Casella TX 12 portable gas detectors) measure each subject's exposure level over a 24 hour period. Each subject also completes an activity diary during the monitoring period and a housing questionnaire. Exhaled breath is also analysed for CO. These measurements will be used to estimate COHb for comparisons between the two groups. So far, not enough data have been gathered on this part of the project to allow meaningful results to be presented.

Results

Hospital study

Data have been collected for 288 cases and 87 controls. Of these, data have been processed for 208 cases and 67 controls. A summary of the COHb and MetHb levels is provided in Tables 2.2.5a and 2.2.5b.

The data have been subdivided into two sets, smokers and nonsmokers, since the overwhelming influence on COHb level is the smoking habit of the subject.

The variance of the data in smokers was such that no significant difference between the well and the vulnerable was demonstrated either for COHb or MetHb. In the nonsmokers, however, the difference although small was significant ($p < 0.001$ for both COHb and MetHb).

The distribution of the two measured parameters was also of interest. While CoHb levels followed a logarithmic distribution skewed to the left, MetHb levels were normally distributed.

Table 2.2.5a A summary of COHb (%) in smokers and nonsmokers in the vulnerable elderly (cases) and the well elderly (controls)

	Smokers		Nonsmokers	
	Vulnerable	Well	Vulnerable	Well
Number	34	9	74	58
Mean	2.0	2.1	1.0	0.8
Median	1.7	1.8	0.9	0.8
Standard deviation	1.3	1.2	0.5	0.2
Inter-quartile range	1.1–2.4	1.1–2.7	0.7–1.2	0.7–1.0

Table 2.2.5b A summary of MetHb (%) in smokers and nonsmokers in the vulnerable elderly (cases) and the well elderly (controls)

	Smokers		Nonsmokers	
	Vulnerable	Well	Vulnerable	Well
Number	34	9	174	58
Mean	0.6	0.5	0.6	0.4
Median	0.5	0.5	0.6	0.5
Standard deviation	0.1	0.1	0.2	0.1
Inter-quartile range	0.5–0.6	0.4–0.6	0.5–0.7	0.4–0.5

Discussion

While interesting, these interim results do not necessarily indicate the final conclusion of the study since the full number of subjects has not been studied. The observation of slightly higher COHb levels in elderly people who are emergency admissions to hospital may indicate their increased exposure to CO but it may also indicate a difference in their endogenous CO metabolism. Also, these data still need to be analysed for age and gender effects. The interpretation of higher MetHb levels in the more vulnerable elderly at this stage of the study is similarly constrained.

While it is unsurprising that the COHb level is higher in smokers than in nonsmokers, it is surprising that MetHb levels were the same since cigarette smoke contains NO. The paradox, however, has been noted before by Borland *et al.* (1985) who calculated that the effect of NO in cigarette smoke was more than one order of magnitude less than that of the CO present.

Reference

Borland C, Harmes K, Cracknell N, Mack D & Higgenbottam T (1985) Methaemoglobin levels in smokers and non-smokers. *Arch Environ Health, 40*, 331–333

2.2.6 Distribution of carboxyhaemoglobin levels in British men aged 60–79 years

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March 1998–February 2000; Second Joint Research Programme

Introduction

The level of blood COHb is a marker of important sources of indoor air pollution, including cigarette smoking and use of gas combustion appliances for cooking and heating, particularly when ventilation is inadequate. A high level of COHb, as well as being a direct marker of toxic effects, may also have an independent relationship to risk of cardiorespiratory disease, although this remains controversial.

The British Regional Heart Study provides a nationally representative framework in which the levels, determinants and prognostic implications of COHb levels can be examined. In 1978–1980, a stratified random sample of 24 towns (50 000–125 000 population) in England, Wales and Scotland was selected, ensuring representation of all major regions. A random sample of 300 men aged 40–59 years was drawn from a single socially representative general practice in each town. In all, 7735 men (78% response rate) had a detailed assessment including a questionnaire on personal and family factors, an electrocardiogram, lung function tests and a blood sample for biochemical and haematological measurements.

All men have been followed for all-cause mortality at the national central registers in Southport and Edinburgh and for fatal and nonfatal cardiovascular events via General Practice. Remeasurement of all surviving study men in 22 of the 24 study towns is being carried out during 1998 and 1999, when they will be aged 60–79 years (n = 5800). Carboxyhaemoglobin levels are being measured during this follow-up survey.

Aims

- To investigate the distribution of COHb levels in a nationally representative population of men aged 60–79 years, including seasonal, geographic and social class variation
- To determine the contribution of factors in the indoor environment including cigarette smoking, domestic heating and cooking
- To study the relationships between COHb levels and both prevalent and incident cardiovascular disease

Methods

All surviving 5800 men in the British Regional Heart Study are being invited for remeasurement. A team of three trained survey nurses is visiting each town in turn between February 1998 and December 1999. Each survey visit takes 2 weeks and follows the same order as in the earlier survey, which ensured that towns with similar geographic and mortality characteristics were not examined together. The assessments made include questionnaire information on health, lifestyle factors, housing and social factors. Physical measurements are being made and a venous blood sample collected. A 1 ml sample of whole blood in fluoride oxalate is being transported overnight to a single central laboratory (Dr S MacFarlane, Whittington Hospital, London) for analysis of COHb within 36 hours of collection. COHb is being measured using a co-oximeter (AVL Medical Instruments, Ltd; coefficient of variation at COHb of 2.0% = 0.05). A full blood count, biochemical profile, and haemostatic and rheological factors are also being assessed. Replicate measurements are being made in 5% of all study participants.

Preliminary results

After measurement of subjects in ten study towns, a total of 1800 subjects has been measured (78% response rate). The distribution of COHb levels, as expected, shows a strong positive skew (median 0.4%, mean 0.8%, SD 1.1%, range 0.0–7.4%). Detailed analyses of the determinants of COHb will be carried out once data collection is completed in December 1999.

2.3 Relationship between indoor and outdoor levels of air pollutants

2.3.1 Indoor/outdoor relationships of benzene and particulate matter

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November 1996–November 1999; January 1998–July 2000; First and Second Joint Research Programmes

Introduction

In this study, the relationship between indoor and outdoor levels of benzene and particulate matter was examined in a number of domestic homes. The homes were chosen on a location basis but of most interest were those homes with roadside locations, where the penetration of benzene and particulate matter from traffic-related sources could be investigated. The chemical composition and mass of particles were examined in order to gain information as to the source of particles found inside the homes.

Sampling methods

Data on near real-time particle mass concentration were obtained with a TEOM, fitted with a PM_{10}, $PM_{2.5}$ or PM_1 inlet. It was not possible to obtain time-resolved data for chemical and mass analysis for all three particle sizes. Modified Andersen cascade impactors, using a pre-separator (PM_{10}) and a back-up stage were used to obtain information related to the size distribution of particulate mass and composition. Pre-washed QMA filters were used to determine mass concentrations of organic and elemental carbon, metals (lead, iron, zinc and calcium) and water-soluble ions (chloride, nitrate, sulphate and ammonium).

Hourly benzene concentrations were determined by the collection of benzene onto Tenax TA packed sorbent tubes, at a flow rate of 100 ml/min. The tubes were fitted to an automatic multi-sampler that draws air through each tube for 1 hour followed by a 15 min interval, during which the valve changes for the next tube in sequence. Tubes were subsequently analysed by gas chromatography and flame ionisation detection (GC/FID) with an ATD-50.

Identical sampling equipment was deployed inside and outside of the homes over 5 day periods (Monday to Friday). The outdoor unit was positioned to avoid areas of rapidly changing airflow, but at roadside sampling sites, where security considerations were usually greater than at rural locations, the unit was positioned close to the house. The indoor unit was positioned to cause minimal intrusion to the occupants and noise-insulated boxes were used to house the pumps that were required for sampling.

Results and discussion

Benzene

Hourly average concentrations of benzene were collected inside and outside six domestic homes. The ambient levels of benzene measured at urban roadside locations were greater than those measured in rural locations. Elevations in outdoor benzene were associated with road traffic but such elevations were rarely found to be reflected in the indoor data.

Particulate matter

Mass concentration of particulate matter was monitored at seven homes, two of which were sampled more than once. Compositional data of the three size fractions were obtained, using modified Andersen cascade impactors, from five locations.

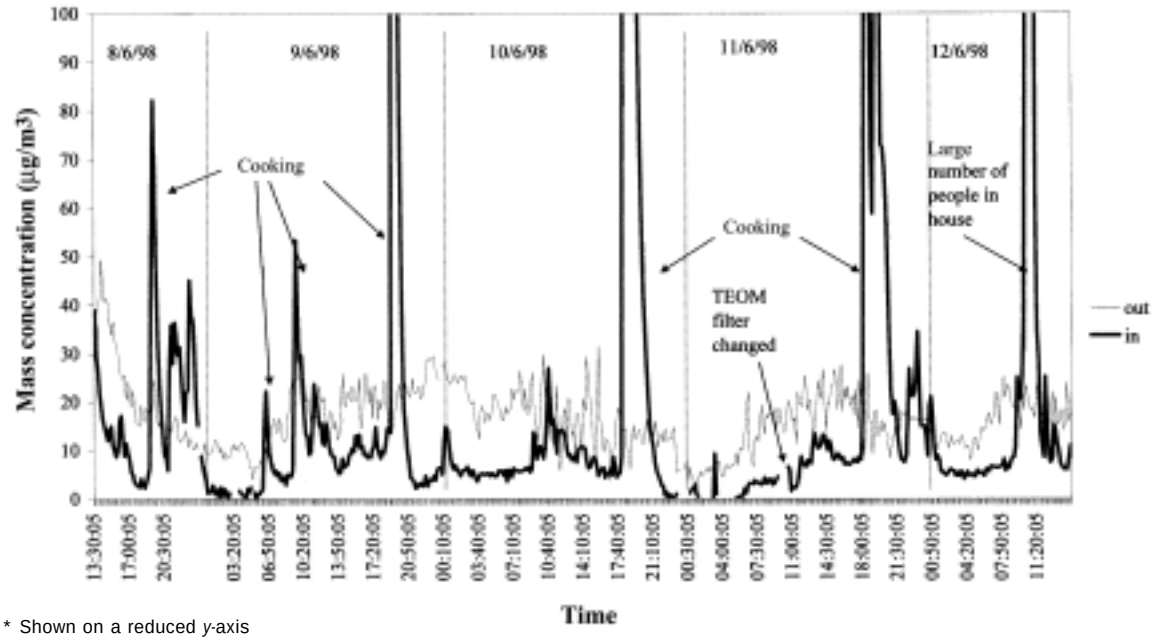
Figure 2.3.1a shows a TEOM trace from a house at a typical roadside sampling location. The house was occupied by six students. Diurnal trends in the outdoor data can be seen, with increases in PM_{10} at times associated with increased traffic. Concentrations inside the home were not correlated with those measured outside. Increased concentrations of PM_{10} inside were associated with times when the occupants got up in the morning and made breakfast and when the evening meals were cooked. Concentrations of PM_{10} inside the

house tended to be lowest during the night-time and greatest during times of cooking. Indoor/outdoor (I/O) ratios, calculated throughout the day (Table 2.3.1a), also showed an increasing influence of human activity on PM_{10} mass concentrations.

Analysis of the particles indicated that, while there was a greater mass of chemical species measured outdoors, there was a greater proportion of fine particles inside the home. These fine particles were composed mainly of organic carbon, sulphate and ammonium. Iron, in coarse particles, was the predominant metal species that was measured, while lead and zinc were present mainly in fine particles.

From other sampling sites, a better correlation between indoor and outdoor concentrations of PM_1 and $PM_{2.5}$ could be seen. The presence of people was shown to have very little influence on the generation of PM_1, an increased influence on $PM_{2.5}$ but a much greater influence on the generation of PM_{10}. A sampling site in a rural location showed that, while the presence of a person had no observable effect on PM_1 mass concentration, the activity of cooking did generate particles of this size.

Figure 2.3.1a Indoor/outdoor PM_{10} mass concentration measurement* taken at Roadside House 1, 8–12 June 1998



* Shown on a reduced *y*-axis

Table 2.3.1a Mean I/O ratios for PM_{10} throughout the day, at Roadside House 1 (June 1998), calculated from 10 min mean values

Date	Night-time (00:00 to 08:00)	Daytime (08:10 to 17:00)	Evening (17:10 to 00:00)	24 hour (00:10 to 00:00)
8/6/98	–	–	1.5	–
9/6/98	0.4	0.6	1.9	0.9
10/6/98	0.3	0.7	13.2	4.0
11/6/98	0.3	0.4	3.9	1.4

2.3.2 A comparison of outdoor and indoor levels of airborne particles at two sites on Marylebone Road, London*

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Introduction

It is now generally accepted that there are potentially serious health effects (such as shortening of life) among the general public from the inhalation of raised levels of airborne particles in the environment. Currently, environmental airborne particles are monitored using fixed site monitors at outdoor locations, as part of the DETR's automatic urban and rural network (AURN). These sites are normally in centres of high population and the data produced are used to estimate the risk to health for that population. However, with people spending on average over 80% of their time indoors it is important to determine the rate at which outdoor particles penetrate to indoor locations.

To address this problem, the DETR has funded a project (see Section 2.3.1), in which identical TEOM mass monitors were sited inside, and in the gardens of, homes in and around Birmingham. As one of the major outdoor particle sources derives from road traffic, the choice of homes monitored in this study included a number that were sited on busy main roads in the centre of Birmingham. However, it soon became clear that the results were dominated by indoor sources, and locations were required where traffic-generated particles dominated and there were no domestic sources.

The aim of this project is to compare the mass concentrations of particles inside and outside two buildings on the Marylebone Road in Central London.

Methods

Monitoring was carried out in an open-plan rest area, and a small unoccupied office at the University of Westminster (UOW). Both locations were on the second floor with windows opening onto the pavement of Marylebone Road, where positioned some 5 m away is the AURN kerbside site, which provided the outdoor data. At Westminster City Council Offices (WCC), about 300 m to the west, two unoccupied offices were used for the monitoring. One was on the first floor with windows facing Marylebone Road, while the second was immediately below at basement level. The council's own instrument, which was located in a 'well' between the building and the pavement, was used to provide the outdoor data.

A TEOM direct-reading mass monitor was used for the indoor measurements, which were compared with similar data from TEOM instruments located outside as explained above. Measurements were mainly of the PM_{10} fraction, with limited periods of monitoring of $PM_{2.5}$ and PM_1 fractions at UOW and WCC, respectively.

Results and discussion

A summary of the broad trends of the data is given in Table 2.3.2a.

At WCC, there was very little difference in the average in/out ratios for PM_{10} particles, with type of day (weekday or weekend) showing no consistent effect. The average in/out ratios for PM_1 particles were very similar to those for PM_{10}, indicating possibly that, for this building, the fine particles in the PM_{10} fraction dominate behaviour.

At UOW, the in/out ratios for the $PM_{2.5}$ particles are very similar, but there is an inconsistency in the PM_{10} data for the two monitoring periods in UOW1 — the rest area. There is no plausible explanation for this apart from the possibility that the size distribution of the particles increased during the 2 month sampling period.

* Not funded directly as part of Joint Research Programmes

Table 2.3.2a Indoor/outdoor ratios of particulates at two sites in London

Site	Dates	Particle fraction	In/out ratios			Regression factors		
			All data	Week days	Weekends	Slope	Intercept (μg/m^3)	R^2
WCC 1st floor	1–20/4/98	PM_{10}	0.62	0.61	0.65	0.54	2.9	0.80
WCC 1st floor	28/4–5/5/98	PM_{10}	0.64	0.63	0.67	0.53	3.9	0.62
WCC Basement Fan off	3–24/7/98	PM_{10}	0.72	0.72	0.70	0.36	7.8	0.49
WCC Basement Fan off	24/7–5/8/98	PM_1	0.68	0.67	0.69	0.37	5.5	0.58
WCC Basement Fan on	5–13/8/98	PM_{10}	0.69	0.68	0.70	0.58	3.3	0.69
UOW Rest area	5–22/5/98	PM_{10}	0.76	0.78	0.73	0.72	0.5	0.72
UOW Rest area	23/5–24/6/98	$PM_{2.5}$	0.74	0.73	0.75	0.49	4.7	0.44
UOW Rest area	24/6–3/7/98	PM_{10}	0.49	0.46	0.53	0.19	10.3	0.24
UOW 2nd floor	14/8–9/9/98	PM_{10}	0.48	0.47	0.50	0.30	5.0	0.45
UOW 2nd floor	9–25/9/98	$PM_{2.5}$	0.75	0.74	0.80	0.56	3.5	0.72

WCC, Westminster City Council; UOW, University of Westminster

2.4 Modelling exposures to particulate air pollutants

2.4.1 Modelling of indoor and personal exposures to air pollutants

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January 1998–June 2000; Second Joint Research Programme

Introduction

Exposures to air pollution (including particulates) are due to both indoor and outdoor sources and activities. Although data exist on concentrations and exposures in different indoor and outdoor microenvironments, the relative contributions of indoor and outdoor sources to personal exposure have rarely been quantified. We have developed a computer model (INTAIR), coded in Visual Basic, that allows the relative contributions to personal exposure of three components to be quantified: outdoor exposure, indoor exposure due to indoor sources, and indoor exposure due to penetration of outdoor air. The core of the INTAIR model describes the physical processes determining indoor pollutant concentrations as a function of outdoor concentrations: air exchange rates, filtration factors, deposition velocities, room dimensions and indoor emission rates. This physical model is then linked to different time–activity patterns to determine the effects of different sources on the frequency distribution of exposure. In this project, we will develop this model further, test its performance against measurements of indoor and outdoor concentrations, and of personal exposure, and apply it on a national or local scale to specific policy-related issues. As the first stage of this development, the particulate component of the model has been modified to provide size-dependent parameterisation and compared with simultaneous indoor and outdoor measurements of particulates. This abstract reports on the key findings of this component of the research programme.

Methods and results

Comparisons of particle number concentrations in indoor and outdoor locations have been made using Grimm particle counters, which provide continuous data in seven particle size classes in the range 0.75–15 µm, and are thus well suited to testing simulations of temporal variations in indoor and outdoor concentrations for all but sub-micron particles. Measurements show a reasonable agreement between the predicted and observed indoor concentrations in empty offices over periods of 24 hours, while the dynamics of indoor concentrations, with a 'damping' of short-term fluctuations in outdoor concentrations, was well represented by the model. However, occupancy of the rooms resulted in large discrepancies between the simulated and measured concentrations, especially for particles greater than 2 µm in size, even when the level of activity of the occupant was minimal.

A further measurement campaign was carried out in school classrooms, with simultaneous measurements of indoor and outdoor concentrations being combined with a record of activity within the classroom. Large peaks in indoor concentrations, coinciding with peaks of child activity within the classroom, were observed in certain schools; the rates of decay of these peaks provide *in situ* data on the deposition rates of particles of different size classes. Additional tests have been carried out to attempt to isolate the factors associated with the increased indoor concentrations in occupied rooms.

Further validation of the model for particulates is planned in home environments, in collaboration with Dave Mark at Birmingham University, who is making simultaneous indoor and outdoor measurements using TEOMs in homes in and around Birmingham. Field measurements have been carried out to characterise the air exchange rate of six of these homes, prior to more detailed analysis of the measurement data in early 1999.

Discussion

The data obtained to date indicate that, although the physical model provides satisfactory predictions in empty rooms, it fails to account for the effects of room occupancy and activity. Although the difference in

measured concentrations of particulates between personal monitors and co-located microenvironmental monitors is well-established, and has been commonly attributed to a ‘personal cloud’, the nature of the particles, the physical processes involved, and the health significance are all poorly understood. Further tests are planned to address these gaps in knowledge and to assess whether it is possible to incorporate a physical description of the processes involved in the model, or rely on empirical modifications of model predictions to account for the effect.

2.4.2 Modelling 24 hour average exposure to PM_{10}

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*March 1996–March 1999; First Joint Research Programme**

Introduction

As part of a study of the relationship between particulate air pollution and blood coagulation factors (Section 5.3.4), a model has been developed that uses information from time–activity diaries to reconstruct 24 hour average exposure to PM_{10}. The model is based on limited experimental data and a review of the published literature. It has been validated against 24 hour average measurements of personal PM_{10} exposure.

The model

Twenty-four hour personal exposure (E) was defined as being the sum of an individual's exposures in four different microenvironments: outside, inside their home, on transport and indoors other than in their own home. The model is defined as follows:

$$E = E_{out} + E_{in\_home} + E_{transp} + E_{other\_in}$$

where

E = personal exposure (μg/m³) over 24 hours
E_{out} = personal outside (μg/m³) over 24 hours
E_{in_home} = exposure in own home (μg/m³) averaged over 24 hours
E_{transp} = exposure as a result of transport, whether car, bus etc. (μg/m³) averaged over 24 hours
E_{other_in} = exposure in an indoor environment other than the home

Each of these exposures was based on the average PM_{10} concentration (μg/m³) in the microenvironment and the proportion of time spent in that particular microenvironment. The exposure outside was defined as the product of the average daily concentration measured at the local AUN sites, the fraction of time spent outside over a 24 hour period, the location of a subject's home within the city and the relative volume of traffic outside the home. It also included a contribution to exposure resulting from any gardening.

The exposure in the home was based on the average daily concentration measured at the AUN site, the time spent in the home, the penetration of PM_{10} into the home, the estimated air-exchange rate in the room and the likely aerosol deposition rate. The concentrations associated with various activities and microenvironments such as cooking, cleaning, other dusty activities, exposure to cigarette smoke and pets were also considered. These concentrations were modified by whether or not the activity was judged to take place in a subject's near-field, i.e. within 1 m, or in their far-field, i.e. elsewhere in the microenvironment. Exposure due to transport was based on the average daily concentration measured at the AUN site, the fraction of time spent on transport and an adjustment factor that took into account the difference between using transport and being elsewhere outside. Exposure in indoor environments other than the home was based on the average daily concentration measured at the AUN site, an estimate of the fraction of indoor PM_{10} resulting from outdoor sources and exposure from activities carried out in the subject's far-field.

Validation

A PM_{10} sampling head developed by the Institute of Occupational Medicine, Edinburgh, was used to measure personal PM_{10} exposures over 24 hours. The performance of the PM_{10} head was also compared with the accepted fixed-location PM_{10} sampler used in the AUN and these data were used to adjust the fixed-point monitoring data to correspond to the personal monitor.

Personal PM_{10} exposure was measured on 111 occasions in 109 elderly individuals. Using the model, estimates of an individual's exposure to PM_{10} were made from the diary and the AUN data over the same period. Since there were no corresponding AUN data for three subjects and one subject refused to

* Principal Investigator, Professor A Seaton

complete a diary, PM_{10} values were estimated on 107 occasions. The Spearman correlation coefficient between the observed PM_{10}, the estimated PM_{10} and the various components of the estimated PM_{10} are shown in Table 2.4.2a.

Significant correlations were observed between the measured PM_{10} and the model exposure estimate (E) and the model components dealing with exposure outdoors (E_{out}) plus exposure in the home (E_{in_home}). The components of the estimated PM_{10} that made significant contributions, therefore, appeared to be those derived from indoor and outdoor exposure.

Table 2.4.2a Spearman rank correlation between measured PM_{10} and components of the model

Model component	r_s	p value
E	0.40	<0.001
E_{out}	0.26	0.007
E_{in_home}	0.28	0.003
E_{transp}	0.04	0.67
E_{other_in}	0.04	0.65
E_{cook}	-0.13	0.20
AUN monitor	0.04	0.72

Discussion

The model provides a way of improving estimated personal exposure to PM_{10} by combining data from fixed-point monitoring stations and personal activity diaries. For the subjects involved in the validation study, exposure in cars (or other transport), during cooking and from indoor environments other than their homes did not appear to contribute significantly to exposure. However, this might be because these activities made up a relatively small proportion of their day.

2.5 Characterisation of air pollutants

2.5.1 Physicochemical identification and comparative biopersistence of indoor and outdoor airborne particulate matter

KA BéruBé and RJ Richards

Cardiff School of Biosciences, Cardiff University

January 1998–December 2000; Second Joint Research Programme

Introduction

Particles have been collected from homes (non-workplace environments) in urban (heavy traffic) and rural (light traffic) areas to investigate spatial and temporal variations in PM_{10} concentration, size distribution and inorganic chemistry. Special attention was given to factors such as ambient weather conditions and personal activities (smoking, cooking, cleaning), which were recorded in detailed questionnaires each time the house was monitored. The intention was that each home examined was its own 'control' and account was taken of seasonal changes and different sampling locations within the home, together with a concomitant outdoor sample in the immediate vicinity of the home. By including time in the sampling procedure to accommodate household routines, it is intended to relate particle mass, size and number or chemistry to personal activity and/or outdoor sources.

Methods

The monitoring of PM_{10} was undertaken using high volume air samplers operating at pre-calibrated flow rates of 30 l/min and attached to PM_{10} selective-inlet heads. Particulate matter was collected onto pre-weighed polycarbonate filters. Combinations of electron microscopy (EM; transmission and scanning), image analysis (IA) and quantitative electron probe X-ray microanalysis (EPXMA) were used to determine and compare the morphology and elemental compositions of indoor and outdoor airborne PM_{10}.

'Summer' samples of PM_{10} were collected in two urban (Cardiff City) and two suburban (Cardiff) and two rural (Cornwall) homes in the UK. The test homes in each area were classified as follows: (1) nonsmokers; (2) smokers; and (3) clean house, that is no dusting/vacuuming or use of aerosols prior to monitoring. Monitoring of PM_{10} was carried out on 'dry' days. Test homes were routinely monitored indoors for three 8 hour intervals for a total of 24 hours, along with the simultaneous monitoring of PM_{10} outdoors. Filters were then subjected to gravimetric analysis to determine the mass of PM_{10} collected. The 8 hour time-weighted average (TWA) and 24 hour total mass were calculated and from these values, the micrograms PM_{10} per cubic metre of air for each collection location were determined. In addition, various ratios such as the outdoor:indoor PM_{10} mass were also assessed. Other information on ambient conditions such as weather, and cooking and cleaning habits were also taken into account as a means of source apportionment. After gravimetric analysis, samples were examined by EM to identify the types of particles present on the filters (Figure 2.5.1a), measured for classification of their physical properties via IA, and analysed using EPXMA to determined their chemical composition (BéruBé *et al.,* 1997; Murphy *et al.,* 1998; BéruBé *et al.,* 1999)

Results and discussion

The indoor to outdoor ratios of PM_{10} mass (24 hour total) were calculated from the gravimetric analysis of PM_{10} filters from summer (June to September) collections at urban, suburban and rural test homes. The most noticeable ratios were seen in the urban and suburban homes of nonsmokers, where there was a higher mass of PM_{10} outdoors, i.e. the ratios were greater than one, when compared with the environment indoors. In contrast, the smokers' homes from urban and suburban locations were found to have a higher mass of PM_{10} indoors when compared with the PM_{10} outdoors. Other factors aside from smoking appeared to have driven these ratios below one, i.e. where outdoor air equals indoor air, these being road traffic in the former and DIY in the latter. The comparison of the samples from urban/suburban homes with the two samples from the rural homes showed that the outdoor environment was 'cleaner' than the indoor environment.

The examination of PM_{10} filters by EM revealed a range of different types of particulate matter, as shown in the examples provided in Figure 2.5.1a. These field emission scanning electron microscopy (FESEM) images of rural and urban outdoor/indoor PM_{10} show that the samples are not dominated by a single type of particle but are characterised by several categories of particle. For example, in the urban outdoor environment, sea salt crystals, smelter and soot particles, plant and insect material, along with mineral particulate matter such as gypsum and feldspar, and fibres were regularly encountered. These same types of particles were also found indoors in the urban test homes. In the rural environment, sea salt crystals, minerals and some soot particles were observed outdoors as well as indoors.

Conclusions

The preliminary data on summer collections of outdoor/indoor PM_{10} from urban and rural locations suggest the following: (1) there are greater masses of PM_{10} indoors; (2) indoor PM_{10} are predominately generated by anthropogenic activities; (3) indoor PM_{10} are also derived from the ingress of contaminated air from outdoors; (4) the mass of PM_{10} collected is characterised by seven different types of particle.

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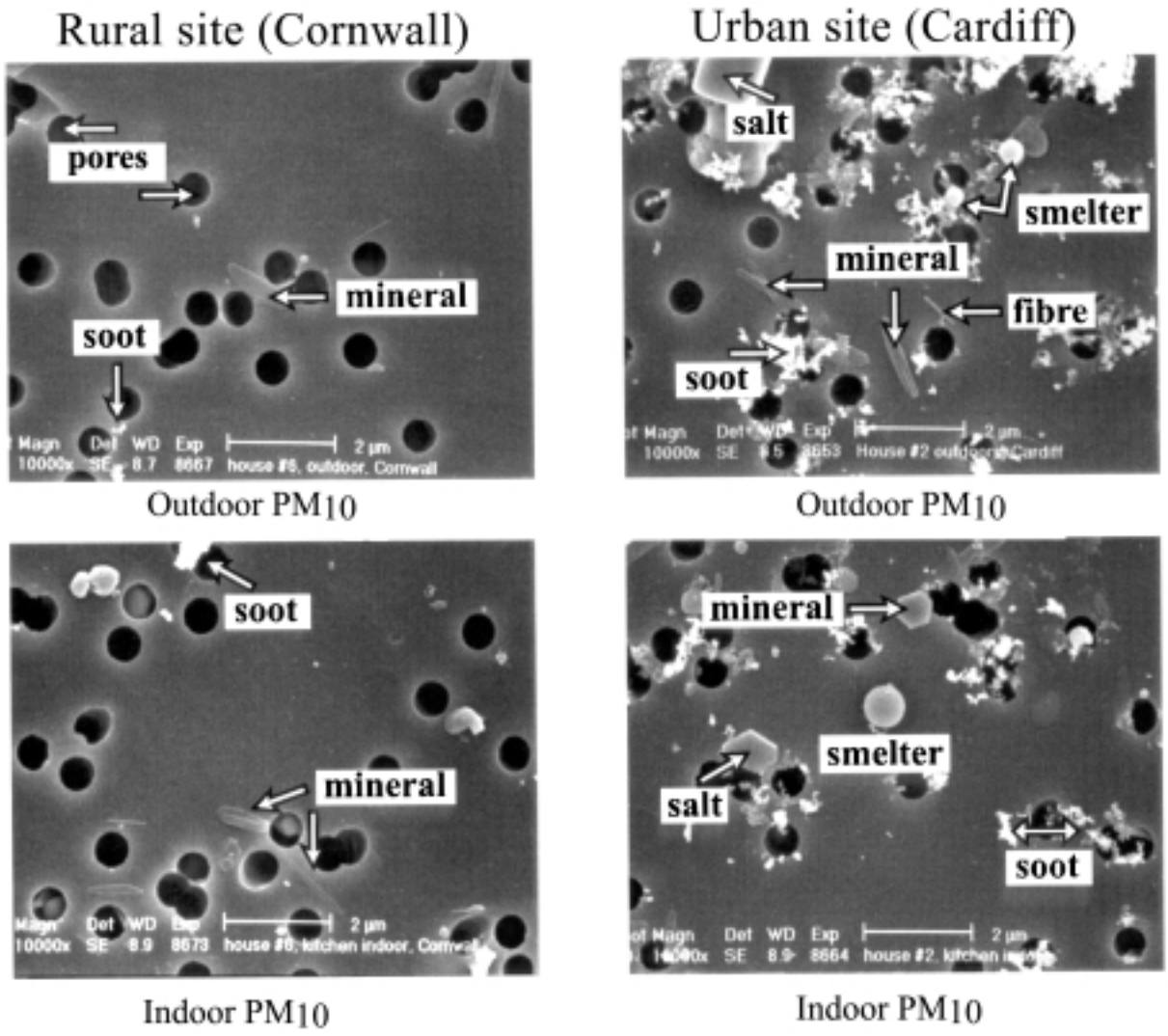
Murphy SA, BéruBé KA, Pooley FD & Richards RJ (1998) The response of lung epithelium to well characterized fine particles. *Life Sci*, *62*, 1789–1799

Figure 2.5.1a Field emission scanning electron microscopy of rural and urban PM_{10}



2.5.2 Airborne particulate pollutants in the South Wales region*

T Jones and R Richards

School of Biosciences, Cardiff University

The study

This project is collecting and undertaking detailed physiochemical analysis of PM_{10} from four sites in the South Wales region. Information is being provided on the heterogeneity of PM_{10} at the four sites, and the mass of the particles collected is being linked to their size distribution. Chemical analyses will assist with source apportionment, such as traffic, industry, mining and agriculture. Characterised samples of PM_{10} will be used in toxicological studies to determine whether the particles cause lung inflammation, increase lung permeability or initiate epithelial damage. A comparison of the results of these studies with previous findings from other environmentally and occupationally derived fine particles, with established safety levels, will permit the end-users to assess risks and supply public advice.

The sites

Nant Helen is an open-cast mining site, operated by Celtic Energy, located to the west of Merthyr Tydfil. Previous epidemiological studies have raised concern that there might be health problems associated with open-cast mining (e.g. Glyneath in Wales, 1997). However, validation of the findings of these investigations is impossible owing to the lack of adequate monitoring, characterisation, and source apportionment of the local PM_{10}. We will monitor particle levels and their characteristics when the pit is operating and producing coal at its planned normal capacity, and also when the pit is engaged in maintenance or reconstruction activities.

Port Talbot is a site that is expected to generate a complex mixtures of particulates, including marine salt particulates, various different heavy-industrial particulates, and PM_{10} from the M4 motorway and other modes of transport. The unravelling of the expected 'cocktail' of different particulates from this location is fundamental to our basic philosophy that, along with measuring the 'mass' of PM_{10}, it is essential to establish particulate size, shape, chemistry and toxicity.

The city of Cardiff, with 300 000 inhabitants, already forms part of the UK PM_{10} monitoring network. The recommended mass standard for PM_{10} is exceeded a number of times each year (QUARG, 1996), but it is unclear whether or not this creates excess health risk. Preliminary studies indicate that within the environs of the city a major component of the PM_{10} could result from traffic activity and, particularly, production of soot from diesel engines.

A rural location in the Black Mountains will be selected that is located a significant distance from the sea, industry, open-cast mining, dense human population and major roads. This rural site will act as a control and for comparisons with the results from the other three locations.

Methods

Particles are collected using high-volume air samplers operating at pre-calibrated flow rates of 30 l/min, attached to PM_{10} selective-inlet heads. The air pumps are powered by a series of heavy-duty 12 V, 35 A batteries, and are therefore nonpolluting. These batteries are sealed, lead-acid, gel (non-spillage), cyclic pump units, and are re-chargeable. Samples are collected onto polycarbonate filters (Millipore DTTP filters, pore size 0.67 µm), as these have proven to be the most durable during sonication to remove particles for subsequent investigations. These chemically inert filters are ideal for particle analysis by electron microscopy. Filters are weighed pre- and post-collection and PM is removed from filters for analysis by our standardised procedures. Simultaneous monitoring of atmospheric conditions during collecting, including temperature, humidity, wind speed and direction, and pollen count, are used to assist with the identification of the particle source(s).

* Funded by the Natural Environment Research Council (NERC-URGENT, 2nd round)

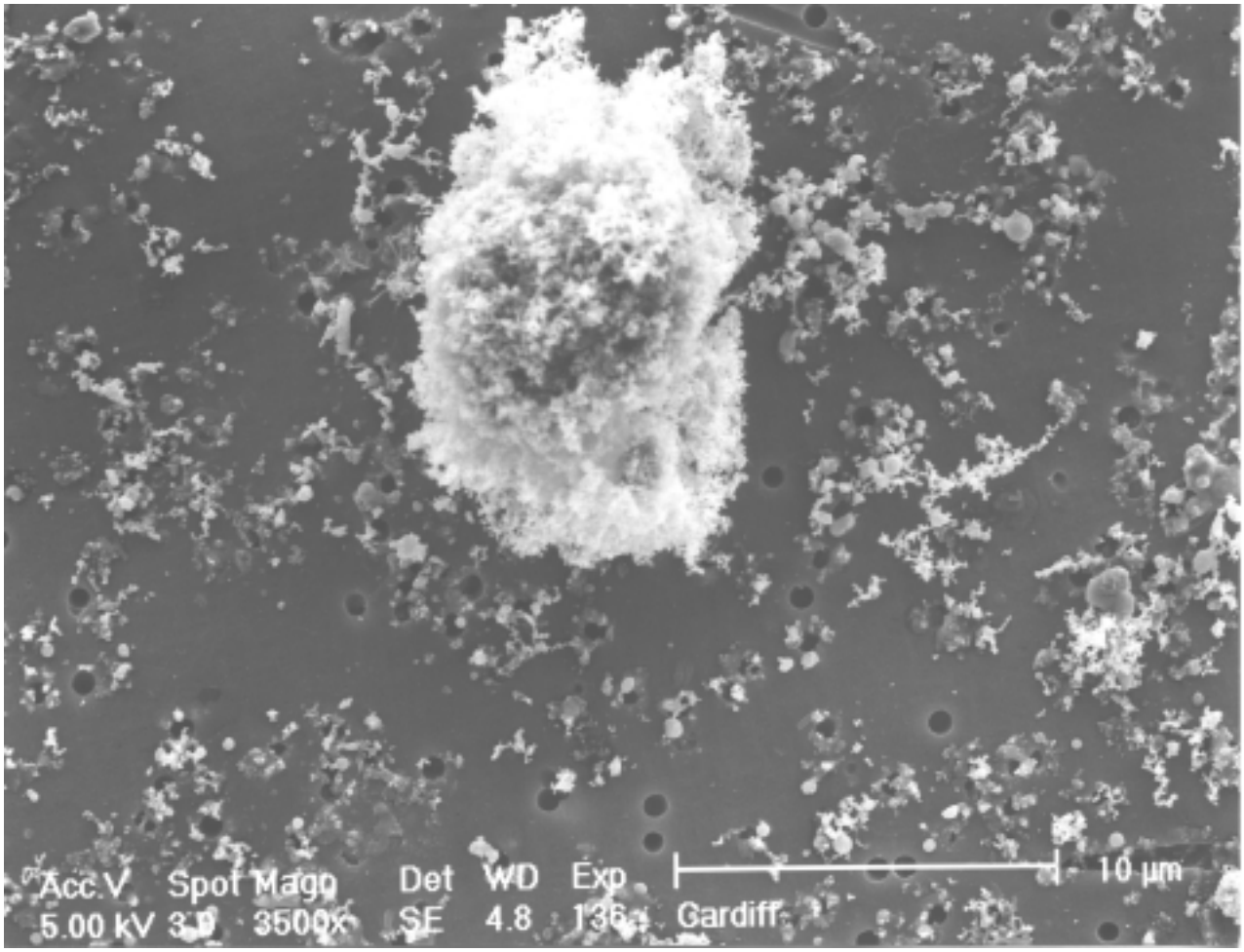
The following methods are used to characterise PM_{10} physically and chemically: transmission electron microscopy (TEM), EPXMA, IA, FESEM, and inductively coupled plasma-mass spectrometry (ICP-MS). EPXMA will be used to determine the chemical composition of the surface and internal regions of fresh and resin-embedded particles, respectively. The number and size of particles collected will be resolved using a combination of TEM and IA to establish individual and aggregated particulate size distributions. Other relevant parameters such as particle surface area, volume, density, and number of individual particles per given agglomeration, will be calculated from the primary IA data. FESEM studies will evaluate the three dimensional micromorphology (i.e. surface characteristics) of PM_{10} (Figure 2.5.2a) and permit the examination of water-soluble particles such as sea salt crystals. ICP-MS will provide quantitative data on chemical composition. In the event that the samples contain a significant mineral component, this can be analysed by PSD-XRD (photosensitive detector X-ray diffraction) at the Nant Helen mine.

The project is currently in the first year of three. Toxicological studies will commence at the start of the second year. These will involve intratracheal instillation of PM_{10} into rat lung to determine whether it induces inflammatory reactions, increases lung permeability, or causes damage to the lining epithelial cells of the small airways or gas exchange units. Decisions on the appropriate toxicological controls will be made after the preliminary physicochemical analysis of particles has been undertaken for each site.

Reference

QUARG (1996) *Airborne Particulate Matter in the United Kingdom* (Third Report), Birmingham, Institute of Public and Environmental Health, University of Birmingham

Figure 2.5.2a Field emission scanning electron micrograph showing airborne pollution collected on a polycarbonate filter, Port Talbot, 1999



Particles include a single 'large' soot, along with numerous, much smaller soot particles

2.5.3 The composition of fine particles as a function of size for urban and rural samples*

GE Andrews

Department of Fuel and Energy, University of Leeds

December 1994–March 1996; First Joint Research Programme

Introduction

The chemical composition and size distribution of atmospheric particles were determined at four sites during the period March 1995 to March 1996. The sites were: a roadside and fifth floor location at the University of Leeds; the Leeds AUN site; and a rural site 20 km north of Leeds.

Methods

Weekly samples were taken using Andersen Cascade impactors that collect nine size ranges — eight impactor stages with 50% cut-off diameters ranging from 0.45 µm to 0.9 µm plus a back-up filter representing particles finer than 0.45 µm. The impactors were fitted with preseparators that make them approximately equivalent to a PM_{10} sampler.

In addition to particle mass concentration, analysis was undertaken for anions, ammonium, acidity, metals (Pb, Fe, Zn and Ca), polycyclic aromatic hydrocarbons (PAHs; the first 16 on the US priority list) and in addition information about volatile matter and carbon content was obtained. Not all analyses were attempted each week because the analytical requirements prevent this.

Results

Total mass concentration

There is good agreement in the total mass collected at the three urban sites with the usual order being ground floor>fifth floor ≈ AUN site. The roadside enhancement relative to the urban background is 5–10 µg/m^3 on a weekly average basis but previous work shows enhancements of 25–30 µg/m^3 during weekday daytime associated activity.

Particle size distribution by mass

On average 10–20% of the urban mass concentration is in the finest particles of less than 0.43 µm, 50% in particles less than about 1.5 µm, and 80% of the PM_{10} mass is particles less than 5 µm in size.

Discussion

The rural site has lower concentrations of particles across the whole size range but this is most noticeable for the finest and coarsest particles. Urban aerosols have a higher proportion of vehicular (and possibly industrial) emissions, which contain particles in the very fine size range, compared with the rural site. The rural aerosol is also depleted in the larger particles, which result from the effects of human activity, including road dust raised by vehicular motion, building activity and industrial emissions.

* Completed by April 1998, abstract presented at second annual review meeting

2.5.4 Comparison of gaseous emissions from different types of pillow

D Crump[1] and D Strachan[2]

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September 1998–August 1999; Second Joint Research Programme

For many years patients with asthma or rhinitis have been advised to avoid feather-filled bedding on the grounds that feathers are potentially allergenic and would tend to harbour house dust mites. A number of organisations recommend the use of synthetic materials for pillows and duvets. However, several recent studies have raised the possibility that synthetic pillows might actually increase the risk of asthma (Strachan & Carey, 1995; Kemp *et al.,* 1996; Butland *et al.,* 1997*).*

It has been proposed that the higher risk of asthma could be due to higher levels of allergen in non-feather pillows but Strachan & Carey (1997) showed that the association of asthma symptoms with non-feather pillows was equally strong among children with and without mite allergy, implying that mite allergy is unlikely to be the total explanation. A more speculative explanation for the difference in risk is that synthetic pillows, particularly newly brought ones, might emit irritant gases that exacerbate respiratory symptoms in sensitive individuals. Due to their proximity to the nocturnal breathing zone, even low concentrations of gases emitted from bedding might contribute substantially to cumulative personal exposure.

The emission of VOCs from different types of pillow is being assessed by the BRE using specialised chamber testing facilities. These have been developed at BRE for the study of emissions from building materials, furnishings and consumer products to evaluate their likely impact on indoor air quality. A wide range of products has been studied and results reported in several publications (Crump, 1995; Crump *et al.,* 1997; Yu & Crump, 1998). The emission test facility consists of 1 m^3 stainless steel chambers supplied with conditioned clean air and a controlled air exchange rate and air velocity. The emission from a sample placed in the chamber is characterised by measuring the concentration of VOCs in the outlet air. Smaller samples can be examined in 2.4 l microchambers developed at BRE. The VOCs are measured by collection onto adsorbents and analysis by thermal desorption–gas chromatography with mass spectrometry. Reactive aldehydes are determined by a separate technique involving derivatisation before analysis by high performance liquid chromatography.

The first part of the programme has involved the use of 1 m^3 environmental chambers to determine emissions from three types of whole pillow (latex, feather and polyester fibre). This investigation has shown differences in the emissions from the different pillow types. Microchambers are being applied to the examination of a larger number of new pillows. Patients and members of staff at St George's hospital have been supplied with air samplers to place near their bedside. At the end of the monitoring period the patients will give their pillows to BRE, in exchange for new pillows, and BRE will undertake emission tests on these for comparison with results for new pillows.

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3 Mechanisms

3.1 Particles

3.1.1 The mechanism of ultrafine particle damage to respiratory epithelium*

SA Murphy, KA BéruBé and RJ Richards

School of Biosciences, Cardiff University

May 1996–May 1999; First Joint Research Programme

Research objectives

Toxicological and mechanistic studies of particulate matter of <10 µm aerodynamic diameter (PM_{10}) and its major UK urban component, diesel exhaust particles (DEP), on epithelial lung cells were carried out, specifically to:

- determine the importance of particle mass, size, surface area and surface chemistry using *in vitro* techniques;
- determine whether epithelial cells (Clara and type 2) accumulate DEP and whether ageing of DEP post-collection is important in toxic reactions;
- examine the cellular reaction, bioreactivity and fate of small masses of DEP in comparison with other fine particles upon instillation *in vivo*; and
- provide the methodology to examine the toxicology of PM_{10} samples from the UK automatic urban network (AUN).

Particle characterisation

The importance, difficulty and time requirement for this facet of the work were underestimated at the outset. Individual DEP (spherulites) of 30 nm readily aggregated such that every sample prepared for toxicological studies comprised a heterogeneous physicochemical mixture (Figure 3.1.1a). Transmission electron microscopy (TEM) was used to determine the number of particles of a given size, together with their calculated mass and surface area in a sonicated sample of DEP (Figure 3.1.1b). The 10% of particles that are ultrafine (<100 nm) have very little mass. In contrast, over 45% of the mass is accounted for by a small number of coarse (>2.5 µm) particles. Sonicating collections of DEP (the best method for dispersal for *in vitro* and *in vivo* studies) changes the size distribution (Figure 3.1.1c). Sonicated DEP particles had a smoother surface, a reduced ultrafine component and greater aggregation than impacted (untreated) counterparts. Furthermore, using electron probe X-ray microanalysis (EPXMA), some redistribution of surface or internal elements was found in DEP that had been sonicated. The implications of these findings for toxicological studies and further information on the metals associated with the surface and internal cores of DEP are reported by BéruBé *et al.* (1999). Further complexity was discovered when commercially available 'ultrafine' carbon black samples were examined; these also have a wide distribution of particle size (aggregations) and different elements associated with their surface and internal cores that, from X-ray diffraction studies, could be linked to the mismatching of graphite crystalline structure. All four carbon black samples showed physicochemical differences but one, M120 (individual spherulite size of

* We acknowledge funding by the MRC (G9527916) and Cardiff School of Biosciences (KAB). Thanks also to Debbie Symonds (Biosciences) and Dr A Gibbs (Llandough Hospital, Cardiff) for help with light microscopy and EM studies

Figure 3.1.1a The structural heterogeneity of DEP as determined by light microscopy

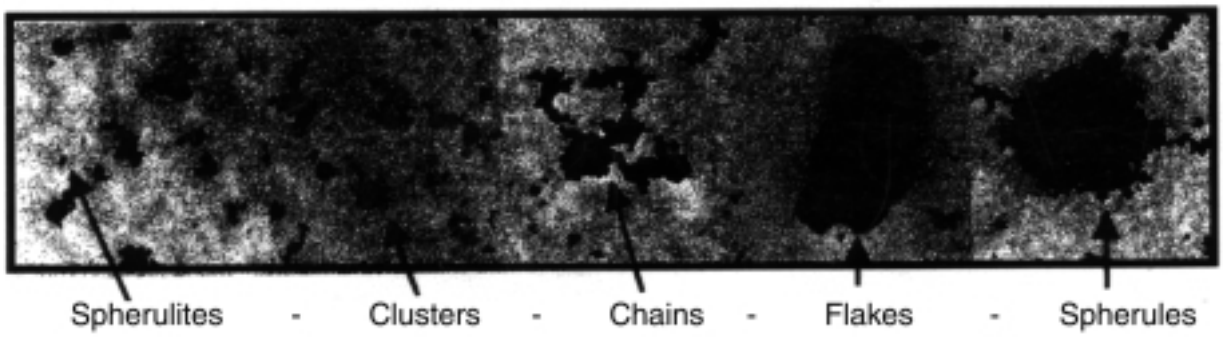
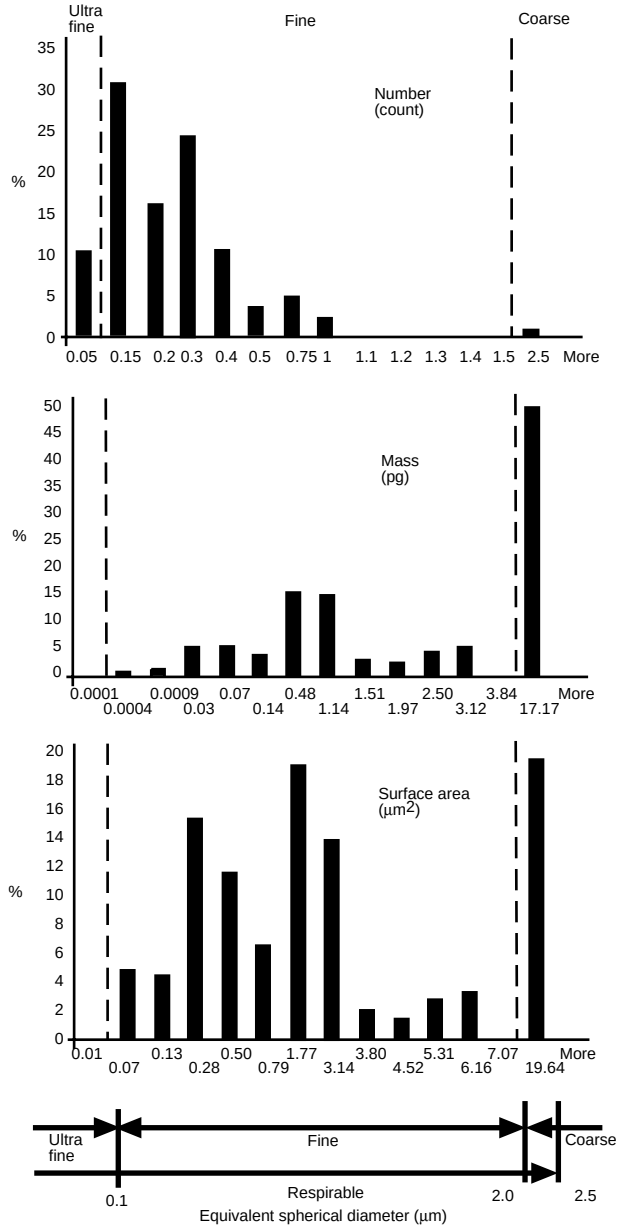


Figure 3.1.1b Distribution of particle number, mass and surface area for a DEP sample as determined by TEM



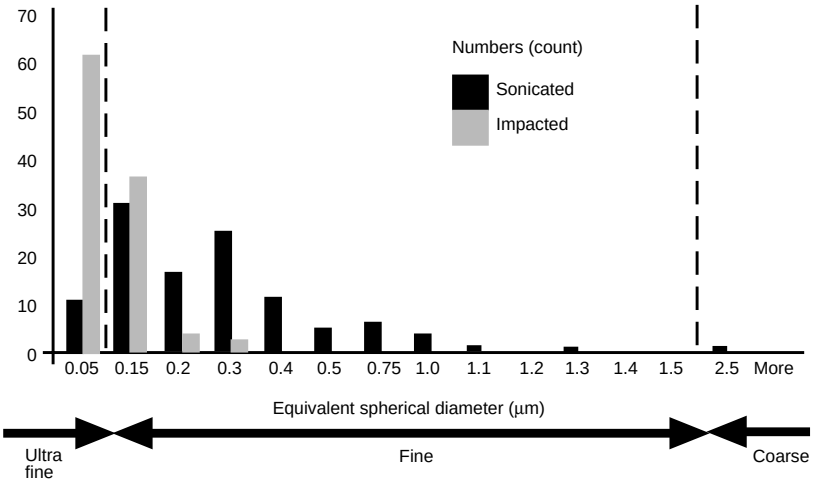
50 nm), was selected for most toxicological studies because, like DEP, it readily forms aggregates but has a much simpler surface chemistry (few associated elements). Other particles that were also fully characterised and used in comparative studies included Cabosil (a pure amorphous silicon dioxide (SiO_2), spherulite size 7 nm, which readily aggregates and, like DEP, has 70% of aggregates of <300 nm in size) and DQI2 quartz (an acid-washed pure crystalline SiO_2) of known high toxicity and lung bioreactivity.

In vitro studies

Fresh isolates of primary rat alveolar type 2 (surfactant producing) and mouse bronchiolar Clara cells were exposed to a variety of doses (by mass) of four carbon blacks and DEP (different samples or the same sample that had been allowed to age after collection) and the interaction assessed by a simple toxicity assay (attachment efficiency to an extracellular biomatrix (basement membrane) over 24 h *in vitro*). The major findings were as follows.

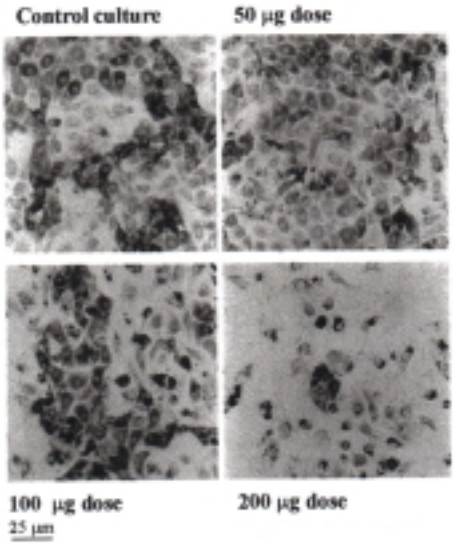
- Carbon black samples exhibited differential toxicity, which was dependent on surface area (calculated from N_2 adsorption studies by the manufacturer). Thus, the smaller the size (diameter) of the individual particle (spherulite) and the larger the available reactive surface area, the greater the toxicity of the particle.
- The most toxic carbon blacks also showed a greater variety of elements on their surface, a feature they shared with DEP.

Figure 3.1.1c Particle number distribution of impacted and sonicated DEP samples



- Four different collections of DEP proved similarly toxic to both cell types, and ageing DEP gradually became less toxic upon storage, with type 2 cells proving more sensitive than Clara cells.
- Confocal and light microscopy both demonstrated that some cells (probably those that are functionally less competent) readily internalise and accumulate the particles or aggregates *in vitro* (Figure 3.1.1d; Murphy *et al*., 1999).

Figure 3.1.1d Clara cells in culture following 24 hour exposure to DEP



Light microscopy of cells stained for nitroblue tetrazolium NADPH-dependent reductase

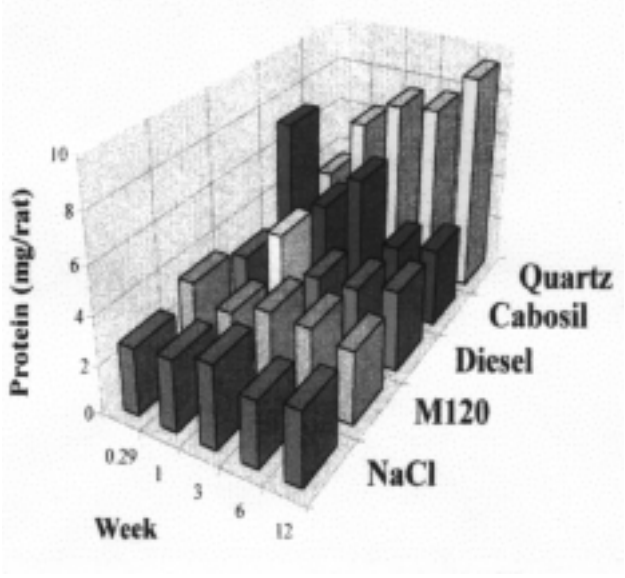
In vivo studies

Small masses (1 mg, chosen because at this lung loading in rat the material is considered to enter the interstitium and produce inflammation) of carbon black M120, DEP, Cabosil and DQl2 quartz were instilled into anaesthetised rats. The animals were sacrificed at different times post-instillation to study early, transient or progressive changes in inflammation, lung permeability (Figure 3.1.1e) or epithelial cell markers and secretions (Murphy *et al*., 1998). Detailed light and EM studies were conducted on lung tissue and lymph nodes (Figure 3.1.1f) of animals exposed to DEP.

The major findings were as follows.

- DQ12 quartz, despite having a minimal ultrafine component, produces progressive lung damage characterised by severe inflammation, persistent increase in permeability, marker enzymes in lavage, mild lipoproteinosis and lymph node enlargement.
- Cabosil, with a high ultrafine component (individual particles 7 nm) and a pure amorphous SiO_2 surface rather than a crystalline one (as with quartz), produces transient changes only, which culminate in a mild inflammation (macrophage influx).
- Carbon black M120 (50 nm particles that aggregate and have a near perfect carbon/oxygen surface) produces no changes in the lung despite being progressively distributed from the alveolar surface, through the interstitium to the lymph nodes.

Figure 3.1.1e Graphical representation of lung surface protein in lavage of animals dosed with different dusts measured at various times post-dose



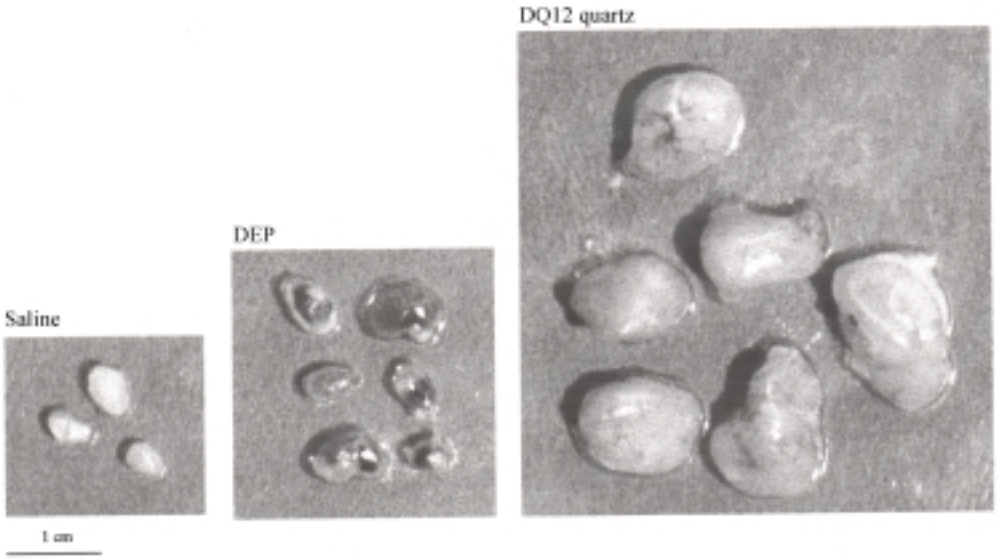
Quartz-dosed animals show progressive damage, Cabosil-dosed animals show transient damage, and carbon black- and DEP-dosed animals show little change relative to controls

- DEP (30 nm particles that aggregate and have a spectrum of elements on their surface) are more reactive than M120, but only marginally so, with evidence of a mild inflammatory effect and possibly some alveolar (epithelial) thickening at 1 and 3 weeks, which resolves by l2 weeks post-instillation.
- There is a slow removal of DEP from the epithelial lining fluid via interstitial cells, which accumulate in large groups around unidentified 'white holes' in alveolar septa. The lung and its cells tend to aggregate DEP and this process is continued in lymph nodes such that, at 6 months post-instillation, large (extracellular) collections are found in the hilar regions. There is no evidence of any size increase or pathological changes in these nodes.

Work in progress

- Increasing assay sensitivity (mostly on lining fluid antioxidant status) to determine whether carbonaceous particles produce effects
- *In vitro* toxicity studies with particles of altered surface chemistry
- Effects of DEP on compromised lungs
- Studies with radiolabelled DEP

Figure 3.1.1f Lymph nodes of animals dosed with saline, DEP or DQ12 quartz, 12 weeks post-dose



Comment

As particles, particularly PM_{10} and DEP, represent such a heterogeneous physicochemical mixture, which may be further altered by processing after collection, it is recommended that characterisation of all samples is important before mechanistic or toxicological studies are carried out. At present, it is not possible to give a clear resolution of the particle characteristic that is most important in cellular damage, if indeed only one characteristic is important. The cell culture studies with carbon blacks emphasise the importance of a small primary particle (size) with a large surface area with localised bio-reactive elements. The animal studies indicate that a small (ultrafine) component is not essential for progressive lung damage and, indeed, a fine carbon black sample with a simple surface chemistry can pass through the respiratory epithelium without causing damage. The crystallinity of the particles (DQI2) or bio-reactivity of a large SiO_2 surface area (Cabosil) is important for progressive and transient damage, respectively. In conclusion, fine-sized carbonaceous components of PM_{10}, including complex DEP, exhibit low bio-reactivity when deposited as small masses in healthy lungs.

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3.1.2 Diesel exhaust particle graveyard*

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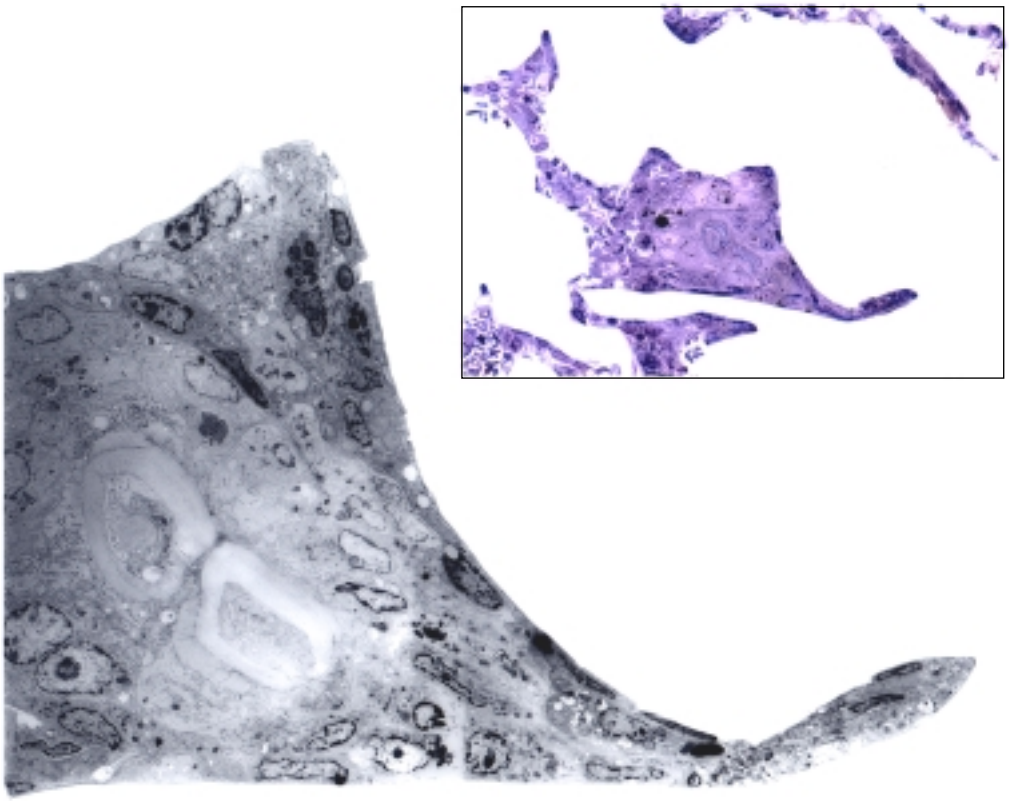
May 1996–May 1999; First Joint Research Programme

As part of a long-term study of the fate of small masses of DEP in the lung (Section 3.1.1) it has been noted that at 6 weeks post-instillation focal accumulations appear in swollen alveolar septa (toluidene blue-stained thin section shown in Figure 3.1.2a). What appear to be DEP, especially if aggregated in a large mass, are just visible in this section.

In order to confirm the presence of DEP and the cells/components in which they were found, an EM montage of the complete alveolar septum was constructed (low power and high power micrographs). A jigsaw (Figure 3.1.2a) was constructed from approximately 100 photographs of the 50 nm thick section of alveolar septum that contains some 80 DEP aggregates (in the region of 4000 individual DEP).

The central area of the swollen septum has two translucent regions, as yet unidentified, in which, or overlying which, are two badly damaged cells containing DEP. Surrounding the central region are a number of interstitial cells containing DEP. Mature Type 2 cells, enriched with lamellar bodies, and Type 1 cells seem to be devoid of particles. However, on the right of the micrograph, lining cells, which appear to be epithelial in origin, do contain DEP. One interpretation is that the central area provides the access to interstitial lymphatic drainage and at this time DEP start to accumulate in thoracic lymph nodes. Can it be that the cells surrounding the central area are awaiting their turn to enter the graveyard?

Figure 3.1.2a Montage constructed from electron micrographs of a 50 nm thick section of alveolar septum containing approximately 80 DEP aggregates



Inset shows toluidine blue-stained thin section

* The financial support of the MRC is gratefully acknowledged

3.1.3 Local and systemic effects of PM_{10} of fine and ultrafine carbon particles

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January 1997–December 1998; First Joint Research Programme

Epidemiological studies have revealed a relationship between the level of PM_{10} and respiratory and cardiovascular mortality. We hypothesise that the ultrafine components of PM_{10} are important in mediating these adverse effects and in determining local lung and systemic effects. We have previously shown that instillation of PM_{10} and ultrafine carbon particles produces lung inflammation and local oxidative stress. In order to test this hypothesis in an inhalation model, rats were exposed to 1000 µg/m^3 of fine (CB, 260 nm in diameter) or ultrafine (ufCB, 14 nm) carbon black. Inhalation of ufCB, but not fine CB, for 7 hours produced a small but significant neutrophil influx (Figure 3.1.3a) Inhalation of ufCB also resulted in an increase in epithelial permeability, as measured by protein in the bronchoalveolar lavage (BAL) fluid, peaking at 16 hours. Lung glutathione (GSH) in rats inhaling ufCB showed a $23.6 \pm 5.4\%$ reduction compared with controls, while at 16 hours total lung GSH of ufCB-exposed rats had increased by 71.3% ($p < 0.01$) compared with controls. By contrast, inhalation of CB did not significantly alter total lung GSH. The antioxidant capacity of plasma was significantly decreased in ufCB-exposed rats, but was unchanged in the CB-treated group, compared with controls. A similar reduction in antioxidant capacity was shown following instillation of PM_{10} (125 µg) (Figure 3.1.3b).

Furthermore, inhalation of ufCB, but not CB, in the regimen described above caused increased pro-coagulant activity in the blood, as shown by an increase in plasma factor VIIc measured as a percentage relative to a human pool, from a median of 390 (range 340–500) to 482 (440–580, $p < 0.01$), 16 hours after inhalation of ufCB, which persisted for 7 days. In order to investigate the properties of PM_{10} that could result in this inflammatory and pro-coagulant effect we studied PM_{10} *in vitro* and demonstrated that it had the potential to release reactive oxygen species, particularly hydroxyl radicals, as shown in a supercoiled DNA depletion assay. In addition we studied the effect of PM_{10} in A549 cells transfected with a nuclear factor κB (NF-κB) construct linked to a luciferase reporter. NF-κB is an important oxidative stress-response transcription factor, which regulates genes for many pro-inflammatory mediators such as cytokines. Treatment of A549 cells transfected with the NF-κB promoter luciferase construct showed that PM_{10} activated NF-κB.

These data show that ufCB, a component of PM_{10}, and PM_{10} itself cause lung inflammation and both local and systemic oxidative stress. Inhalation of ufCB also increased pro-coagulant factors in the blood. These studies may help to explain the mechanism of the adverse health effects of PM_{10}.

Figure 3.1.3a Total cell number and per cent neutrophils in BAL after CB inhalation

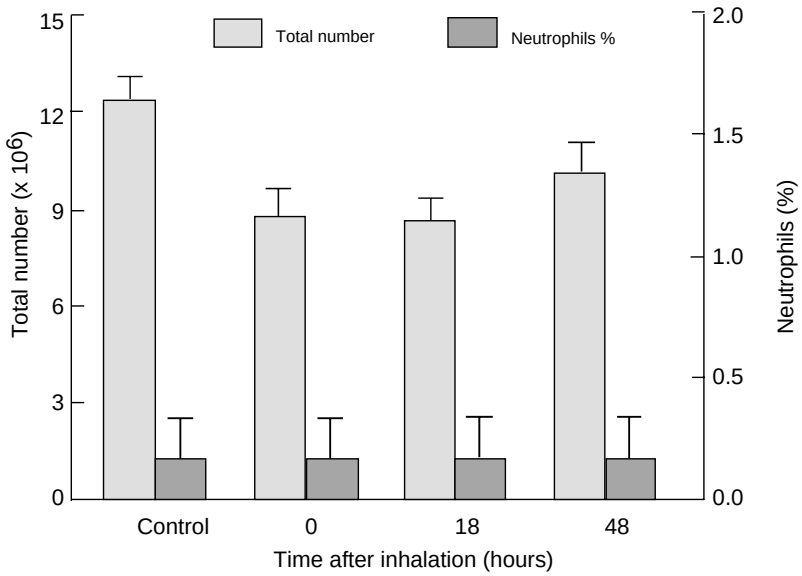
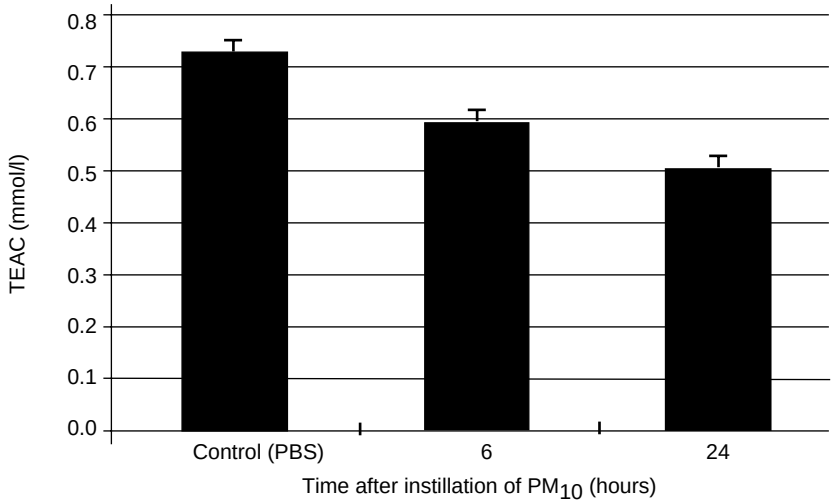


Figure 3.1.3b Trolox equivalent antioxidant capacity (TEAC) in rat plasma at various times after instillation of PM_{10}



3.2 Antioxidant status

3.2.1 Is the response of the respiratory epithelium to ozone modified by the presence of lung lining fluid?

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June 1996–May 1997, First Joint Research Programme

Introduction

This study addresses the hypothesis that the responses of resident inflammatory cells and the respiratory epithelium to the gaseous pollutant ozone (O_3) are dictated, in part, by the composition of the overlying respiratory tract lining fluid (RTLF). We proposed that O_3 is too reactive to cross the RTLF and it therefore initially reacts preferentially with antioxidants present in RTLF. It therefore follows that the antioxidant composition of RTLF can dictate the magnitude of the cellular response to O_3. Moreover, any cellular response that arises following exposure to O_3 is more likely to be due to the presence of secondary products arising from the interaction of O_3 with protein and lipid macromolecules within the RTLF than a direct interaction between O_3 and the respiratory epithelium.

Methods

A novel exposure system was developed that incorporates either rat alveolar macrophages or Type II epithelial cells. Using this system we determined (a) whether overlaying these cells with BAL fluid protects them from O_3-induced damage, and (b) whether the antioxidant composition of BAL fluid determines the degree of protection afforded against O_3.

Results

End-points determined included: (1) Cr^{51} loss from the cells; (2) phosphatase activity; (3) glutathione *S*-transferase (GST) activity; and (4) γ-glutamyl transpeptidase (γ-GGTP) activity. Exposure of Type II cells to O_3 results in the loss of pre-loaded Cr^{51}, alkaline phosphatase activity, GST activity and an increase in GGTP activity. Exposure of alveolar macrophages to O_3 results in the loss of pre-loaded Cr^{51}, alkaline phosphatase activity, GST activity and an increase in GGTP activity.

Exposure of BAL fluid to O_3 for as little as 5 min results in the generation of toxic moieties that injure alveolar macrophages when incubated with them for 2 hours. Injury to Type II epithelial cells is also seen, but only after a 4 hour incubation period.

Discussion

The results obtained suggest that O_3-induced changes in macrophage function and viability, and to a lesser extent Type II cell function and viability, are influenced by the nature of the fluid overlying these cells during the exposure period. In the absence of overlying medium, marked loss of cell integrity was seen following O_3 exposure. In the presence of BAL fluid, cell injury was only seen following the consumption of uric acid and ascorbic acid by O_3. In marked contrast, in the presence of Hanks balanced salt solution (HBSS), little response is seen even following 4 hour exposure to 1 ppm O_3, suggesting that this buffer does not permit O_3 penetration to the underlying lung cells.

Conclusions

Together, these data support the concept that RTLF antioxidants provide the first line of defence against inhaled oxidants. As such, RTLF antioxidant status is likely to play an important role in determining an individual's response to a specific air pollutant.

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3.2.2 A novel exposure system to examine how the impact of airborne pollutants on lung cells is modulated by antioxidants*

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Introduction

In a pilot study (MRC, G9605710; Section 3.2.1) we established a new *in vitro* exposure system to examine the degree of protection afforded by RTLF to underlying macrophage and epithelial cells under oxidative stress. We now plan to use this model to discover the threshold of RTLF antioxidant composition relative to air pollutant (O_3 and NO_2) that precipitates altered underlying cell metabolism. In addition, we plan to determine whether the inflammatory response of the lower respiratory tract to these agents involves release of pro-inflammatory mediators by resident cells. We also intend to establish whether secondary oxidation products are involved in these cellular responses and, if so, whether their effect can be prevented by antioxidant intervention.

Specific aims

The aim is to address the following objectives in relation to exposure of RTLF to O_3 and NO_2 alone, and in combination.

- The relationship between RTLF antioxidant profile, pollutant exposure and the magnitude of the cellular response. In particular, what is the critical threshold of RTLF antioxidants and/or pollutant exposure that triggers altered cell function? Do combinations of pollutants cause a greater, or different, response?
- Which class of secondary (lipid/protein) oxidation product elicits cellular responses.
- Which cells, macrophages or epithelial cells, are most susceptible to modified RTLF. Do the cells release inflammatory mediators that modulate each other or that might be relevant *in vivo*?
- Whether antioxidant supplementation of RTLF provides protection from any detrimental effects of pollutants and, if so, whether the concentrations have therapeutic relevance.

Methods

Rat and human alveolar macrophages and Type II epithelial cells will be examined in the novel exposure system in the presence of autologous RTLF. RTLF will be examined for reduced and oxidised glutathione, uric acid and ascorbic acid. RTLF samples will be analysed for lipid peroxidation products (lipid hydroperoxides) by HPLC. Protein carbonyl concentrations will also be determined. Cell integrity will be assessed by Cr^{51} release and trypan blue exclusion; apoptosis, programmed cell death that will not be detectable by trypan blue exclusion, will be measured, by the TdT-mediated dUTP digoxygenin nick end-labelling (Tunel) method. Cell function will be assessed with measures of alkaline (type II) and acid (macrophage) phosphatase, GST and GGTP in the cell mat, detached cells and conditioned media, as appropriate. The following chemokines/cytokines will be determined: interleukin-8 (IL-8), monocyte chemotactic protein-1 (MCP1), macrophage inflammatory protein-1α (MIP 1α) and RANTES. Also to be determined are levels of tumour necrosis factor (TNF), IL-1 and granulocyte-macrophage colony-stimulating factor (GM-CSF), cytokines that are known to be elevated in inflammatory lung disease. Where changes are significant, mRNA levels (Northern blotting) will be examined to determine whether this reflects transcriptional changes.

This project will commence in August 1999. Preliminary studies have already been performed with human type II cells and O_3 exposure.

* Funded by an MRC Strategic Grant

4 Volunteer studies

4.1 Diesel

4.1.1 Lung inflammation after controlled exposure to inhaled diesel particulates

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July 1997–June 1998; First Joint Research Programme

Introduction

Air pollution from diesel exhaust particles (DEP) is a significant public health concern. Epidemiological evidence suggests a link between morbidity and mortality and increases in atmospheric concentrations of respirable particles. However, the mechanism of effect of inhalation of these particles is unknown. Having developed a system for the controlled delivery of diesel particulates at high ambient concentrations, we studied the inflammatory response in the lungs of normal volunteers.

Methods

Particulate matter was collected from the exhaust of a stationary diesel engine. Ten non-smoking healthy volunteers were exposed for 2 hours at rest to isolated particulates at a concentration of 200 µg/m^3 PM_{10} (particulate matter of <10 µm aerodynamic diameter), or air, in a double-blind, randomised, crossover study. Exposures were followed by serial measurement of spirometry, pulse, blood pressure, exhaled carbon monoxide (CO) levels and methacholine reactivity at 4 hours and 24 hours post-exposure; sputum was induced at 4 hours post-exposure.

Results

There were no changes in spirometry, cardiovascular parameters or methacholine reactivity following exposure to diesel exhaust. There was a significant increase in exhaled CO levels following DEP exposure, maximal at 1 hour (air 2.9 ± 0.2 ppm, diesel 4.4 ± 0.3 ppm, $p < 0.001$). There was an increase in sputum neutrophils at 4 hours following DEP exposure compared with air exposure (air 32 ± 4%, diesel 41 ± 4%, $p < 0.01$). An increase in sputum supernatant myeloperoxidase following DEP exposure was also observed.

Conclusions

Exposure to isolated DEP at high ambient concentrations leads to an identifiable inflammatory response in the lung in normal volunteers.

4.1.2 Airway inflammatory responses to short-term diesel exhaust exposure in healthy and mild-asthmatic subjects

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February 1996–January 1998; First Joint Research Programme

Introduction

We have previously demonstrated that short-term exposure to diesel exhaust at high peak ambient concentration levels (PM_{10}, 300 µg/m^3) induced a marked acute cellular and mediator inflammatory response in the airways of healthy human volunteers, which was accompanied by upregulation of interleukin-8 (IL-8) mRNA in airway tissue and bronchoalveolar lavage (BAL) cells and increased production of IL-8 and Gro-α in airway epithelial cells.

The aim of this study was to evaluate inflammatory responses to a controlled lower dose exposure to diesel exhaust (PM_{10}, 100 µg/m^3) in the airways of healthy and mild-asthmatic subjects. Twenty-five healthy and 12 mild-asthmatic volunteers were randomly exposed to diesel exhaust and air for 2 hours in an exposure chamber on two separate occasions at least 3 weeks apart. Bronchoscopy with bronchial wash, BAL and biopsy sampling were performed 6 hours after exposure.

Results

In the healthy volunteers significant increases in IL-6, IL-8, neutrophils and lymphocytes were observed in the bronchial wash and BAL, while immunohistochemical staining on the bronchial biopsies revealed a significant upregulation in the expression of the adhesion molecule P selectin in the vascular endothelium. There were no differences in the number of neutrophils, lymphocytes and mast cells in the airway tissue. In contrast, no inflammatory responses were noted in the BAL and airway tissue of mild-asthmatic subjects. Similarly, there were no differences in the transcripts of the genes encoding IL-1b, IL-4, IL-5, IL-8, tumour necrosis factor α (TNFα), interferon-γ (IFNγ) and granulocyte-macrophage colony-stimulating factor (GM-CSF) in the bronchial mucosa of mild-asthmatic volunteers.

Conclusions

Short-term exposure to diesel exhaust produces a dose-dependent inflammatory response in the airways of healthy human volunteers. In contrast, the airways of mild-asthmatic subjects appear to be less responsive to exposure to diesel exhaust when inflammatory responses are measured 6 hours after exposure.

4.2 Other pollutants

4.2.1 The effect of a combination of pollutants (NO_2 and O_3) at different concentrations and exposure times on the airway response of mild asthmatics to inhaled allergen over a period of 48 hours

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July 1996–March 1999; First Joint Research Programme

Introduction

We have previously demonstrated that exposure to a combination of 400 ppb nitrogen dioxide (NO_2) and 200 ppb sulphur dioxide (SO_2) leads to increased airway responsiveness of mild atopic asthmatics to inhaled allergen (Devalia *et al*., 1994), and that this effect is maximal after 24 hours (Rusznak *et al*., 1996). The hypotheses behind the present study were that a combination of NO_2 and ozone (O_3) would have similar effects, and that longer exposures to lower levels of the two pollutants would have similar effects to exposures for half the time at double the concentration, thus delivering the same total dose.

Methods

To test this hypothesis, four groups of ten atopic asthmatic individuals were investigated as outlined below.

In Group 1, patients were exposed for 6 hours to air, 100 ppb O_3, 200 ppb NO_2, or 100 ppb O_3 + 200 ppb NO_2, followed by immediate allergen challenge.

Group 2 underwent 6 hour exposures, once to air and on three separate occasions to 100 ppb O_3 + 200 ppb NO_2, with allergen challenge performed immediately, or at 24 or 38 hours after exposure.

In Group 3, patients from Group 1 were subsequently exposed for 3 hours to air, 200 ppb O_3, 400 ppb NO_2 or 200 ppb O_3 + 400 ppb NO_2, followed by immediate allergen challenge.

Patients in Group 4 were exposed for 3 hours, once to air and on three separate occasions to 200 ppb O_3 + 400 ppb NO_2, with allergen challenge performed immediately, or at 24 or 48 hours after exposure.

All exposures were performed in a randomised, single-blind manner in an exposure chamber, with the patients undertaking intermittent moderate exercise. Bronchial allergen challenges were performed with increasing doses of *Dermatophagoides pteronyssinus* allergen (Der p1) from which the number of cumulative breath units (CBU) required to cause a 20% fall in forced expiratory volume in 1 second ($PD_{20}FEV_1$) was calculated.

Results

Results of spirometry performed before and after exposure showed no significant differences between the pollutant exposures to air in Groups 1 and 2. In Group 3, exposure to 200 ppb O_3 and 200 ppb O_3 plus 400 ppb NO_2, but not to 400 ppb NO_2 alone, significantly decreased the FEV_1, compared with exposure to air (mean % change ± SD: +1.92 ± 4.35 for air, vs -3.71 ± 5.17 ($p = 0.013$) for O_3, -4.07 ± 9.18 ($p = 0.05$) for O_3 + NO_2).

In Group 1 the geometric mean $PD_{20}FEV_1$ (log CBU) after exposure to air, 100 ppb O_3, 200 ppb NO_2 and 100 ppb O_3 plus 200 ppb NO_2 was 2.94, 2.91, 2.77 and 2.69, respectively. Differences between exposure to air and pollutants did not reach statistical significance, although a trend was observed for 100 ppb O_3 plus 200 ppb NO_2 ($p = 0.067$).

In Group 3 (involving the same patients as Group 1) exposure to 200 ppb O_3, 400 ppb NO_2 and 200 ppb O_3 plus 400 ppb NO_2 significantly decreased the allergen $PD_{20}FEV_1$, compared with exposure to air (geometric mean: 3.0 for air, vs 2.66 ($p = 0.002$) for O_3, 2.78 ($p = 0.018$) for NO_2, 2.65 ($p = 0.002$) for O_3 + NO_2).

In Group 2, exposure for 6 hours to 100 ppb O_3 plus 200 ppb NO_2 significantly decreased allergen $PD_{20}FEV_1$, immediately, and after 48 hours, but not after 24 hours, compared with exposure to air (geometric mean: 3.12 for air, vs 2.67 ($p = 0.013$) immediate, 2.95 ($p = 0.3$) 24 hour, 2.73 ($p = 0.04$) 48 hour).

In Group 4, exposure for 3 hours to 200 ppb O_3 plus 400 ppb NO_2 significantly decreased allergen $PD_{20}FEV_1$ immediately and after 24 and 48 hours, compared with exposure to air (geometric mean: 3.03 for air, vs 2.59 ($p < 0.0005$) immediate, 2.71 ($p = 0.027$) 24 hour, 2.77 ($p = 0.016$) 48 hour).

Conclusions

For both O_3 and NO_2, significant increases in airway responsiveness to inhaled allergen in exercising mild asthmatics were seen after 3 hours exposure to higher concentrations, but not after 6 hours exposure to lower concentrations. These results suggest that the effects are dependent more upon the inspired concentration (above a certain threshold) than the total inhaled dose of pollutant.

At higher concentrations the effects of the two pollutants were not additive. At lower concentrations (which individually have no effect on either lung function or $PD_{20}FEV_1$), an additive effect was seen; the trend observed in Group 1 was confirmed by a significant result in Group 2 on a different group of patients. This lends support to the theory that the adverse effects of air pollutants occur in combination, and at lower ambient levels than can be demonstrated for individual pollutants in chamber studies.

The adverse effect of this low dose combination appears to be maximal immediately after exposure but still significant after 48 hours, consistent with the hypothesis that both O_3 and NO_2 induce airway inflammation that is sustained for several days after exposure.

References

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Rusznak C, Devalia JL & Davies RJ (1996) Airway response of asthmatic subjects to inhaled allergen after exposure to pollutants. *Thorax, 51,* 1105–1108

4.2.2 The effect of exposure to fine sulphuric acid aerosol on the airway responses to inhaled grass pollen allergen of adults with mild asthma

WS Tunnicliffe[1], D Evans[2], D Mark[2], RM Harrison[2] and JG Ayres[1]

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[2]The Institute of Public and Environmental Health, University of Birmingham

April 1996–April 1999; First Joint Research Programme

Introduction

Epidemiological evidence supports an association between exposure to particulate air pollution and acute exacerbations of asthma. Human challenge studies, including our own, however, suggest little or no direct effect of particulate exposure on the resting lung function of asthmatic adults. Recently it has been recognised that some gaseous air pollutants, including O_3 and NO_2, can enhance the airway response of asthmatics to inhaled allergen and it has been suggested that this might be one of the mechanisms through which they exert their adverse health effects in this group. We have investigated the effect of 1 hour exposures to two concentrations of particulate sulphuric acid aerosol on the subsequent airway response to grass pollen allergen in adult volunteers with mild asthma.

Methods

Volunteers with mild asthma and a positive grass pollen skin prick test underwent a grass pollen allergen (Timothy and Cocksfoot, Bayer) challenge at recruitment to determine their provocative dose of allergen required to produce a 15% fall in their FEV_1 ($PD_{15}FEV_1$). Subsequently they were exposed to air (placebo), 100 µg/m^3 (low dose) and 1000 µg/m^3 (high dose) sulphuric acid aerosol (mass median diameter (MMD) 0.3 nm) for 1 hour, double blind, in random order, at least 2 weeks apart. Exposures were through an integrated head-only delivery system. Approximately 14 hours after each exposure subjects underwent a fixed-dose allergen challenge using their pre-established $PD_{15}FEV_1$. The maximum reduction in FEV_1 (per cent change from post-diluent FEV_1) during the first 2 hours after completion of the allergen challenge was defined as the early asthmatic response. After recovery of FEV_1 towards baseline, any subsequent fall in FEV_1 recorded up to 12 hours later was defined as the late asthmatic response. Matched-pair analysis was used to compare changes in FEV_1 values. Paired *t*-tests were used for significance testing.

Results

Thirteen adults (median age 29 years, 4 male) with mild asthma were recruited. Of these, five were using inhaled steroids (median dose 400 µg/day) regularly. Three subjects failed to complete the study; two withdrew owing to a significant deterioration in their asthma control following their first and second fixed dose allergen challenges; the remaining subject withdrew due to the development of another, unrelated medical condition. Their results are excluded from the data analysis. All exposures were well tolerated and there were no significant changes in FEV_1 or forced vital capacity (FVC) with any exposure (data available on request). The mean early asthmatic response to the fixed-dose allergen challenge following air exposure was -14.1%, following 100 µg/m$_3$ sulphuric acid, -16.7%, and following 1000 µg/m^3 sulphuric acid, -18.4%. The difference between air and high-dose sulphuric acid (-4.3%) was significant (95% CI -1.2 to -7.3%, p = 0.013). The difference between air and low-dose sulphuric acid (-2.6%) just failed to achieve significance (95% CI 0.0 to -5.3%, p = 0.051). There was no significant difference between low- and high-dose sulphuric acid (-1.65%, 95% CI 0.52 to -3.83%, p = 0.13). The mean late asthmatic response following air exposure was -6.0%, following 100 µg/m^3 sulphuric acid, -5.5%, and following 1000 µg/m^3 sulphuric acid, -6.7%. There were no significant differences between these values.

Discussion

These results suggest that, at least at high mass concentration, fine sulphuric acid aerosol can potentiate the early asthmatic response of patients with mild asthma to inhaled grass pollen allergen. The size of the effect is small and its clinical importance is uncertain. It is similar in magnitude to that seen with other, gaseous

air pollutants and is broadly concordant with Bylin and colleagues observations of the enhancement of the early asthmatic responses to birch pollen of asthmatic volunteers exposed to particulate matter ($PM_{2.5}$ >100 µg/m3) and NO_2 (300 µg/m3) in a road tunnel. The effects of lower mass concentrations of sulphuric acid appear equivocal. We failed to detect any significant effect of sulphuric acid aerosol exposure on the late asthmatic responses of our volunteers. While our study design made such a comparison valid, significant late asthmatic responses (>10% fall in FEV_1) were observed in only three of the ten completing subjects following placebo exposure, rendering the study relatively under-powered to examine adequately the effects of exposure on this outcome.

4.2.3 The effect of challenge with fine particulate sulphuric acid and ammonium bisulphate, and sulphur dioxide on indices of heart rate variability in normal and asthmatic adults

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April 1996–April 1999; First Joint Research Programme

Introduction

The mechanisms through which air pollution impacts on cardiovascular morbidity and mortality are poorly understood. We hypothesised that autonomic pathways might be involved and included the measurement of heart rate variability (HRV) as a secondary outcome measure in a study of the effects of sulphuric acid and ammonium bisulphate particulate, and SO_2 challenge in adult volunteers. Other measures included spirometry, minute ventilation and the oro-nasal partitioning of breathing. Spectral analysis of HRV allows the non-invasive quantitative assessment of autonomic activity. Spectral components include total power (TP), and high frequency (HF) and low frequency (LF) power, reflecting total variability, parasympathetic (vagal), and mixed sympathetic and parasympathetic modulation.

Methods

Volunteers wore a Holter recorder (Oxford Medilog 4500) during pollutant exposures, which were head only, 1 hour in duration, double blind, in random order, and at least 2 weeks apart. Exposures were to air (placebo), 200 ppb SO_2, 200 µg/m^3 and 2000 µg/m^3 sulphuric acid (MMD 0.3 µm), and 200 µg/m^3 and 2000 µg/m^3 ammonium bisulphate (MMD 0.3 µm). R-R data were templated and outliers and ectopics were identified and censored. Spectral analysis was performed by discrete Fourier transformation (Hanning window 512 pts) following cubic spline interpolation, trend removal by least squares linear regression and re-sampling at 3.41 Hz. Powers were reported for each 5 min epoch. Mean powers were compared by paired *t*-tests.

Results

Twelve normal adults (median age 38 years, 5 male) and 12 asthmatic adults (median age 34 years, 7 male) were studied. There was no significant change in FEV_1 with any exposure in either group. Similarly there was no observed effect of exposure on volumes breathed for either group, although a small but significant increase in the respiratory rate of the asthmatic group was observed during SO_2 exposure (vs placebo) (mean 16 b/min vs 15.1 b/min, p = 0.04). There were no differences in the mean, maximum or minimum heart rate of either group between exposures.

The HRV data were found to be non-stationary over the 12 five minute epochs of the exposures, with considerably more variability over the first four epochs and more stability over the last seven; comparisons have been restricted to data derived from the last seven epochs. Generally the groups' responses to active exposures differed qualitatively: TP, HF and LF power tended to be reduced in the asthmatic group and increased in the normal group relative to placebo exposure. The effects of SO_2 exposure were the most marked: in asthmatic subjects the mean differences in TP, HF and LF between air and SO_2 were -1021 ms^2 (p = 0.1), -539 ms^2 (p = 0.3) and -572 ms^2 (p = 0.1), respectively. In the normal subjects, the differences were +1730 ms^2 (p = 0.05), +964 ms^2 (p = 0.1), and +437 ms^2 (p = 0.09). Comparison of mean values during exposure between the two groups showed highly statistically significant differences.

Discussion

These results were unexpected and, although they must be treated with caution, suggest that acute pollutant exposures may have measurable cardiac effects mediated through the autonomic nervous system, in the absence of changes in respiratory function. The observed changes in HRV indices were recorded in subjects not believed to be at increased risk of the cardiovascular effects of air pollutants; in groups at higher risk, such effects may reflect a novel mechanistic pathway for the adverse cardiovascular effects of air pollution.

The observation that sulphur dioxide has opposing effects on HRV indices in asthmatic and normal subjects suggests that this agent may cause bronchoconstriction in these groups by differing mechanistic pathways and may possibly suggest an underlying autonomic abnormality in asthma.

5 Epidemiology

5.1 Indoor air pollution and health

5.1.1 A case–control study of the effects of common indoor pollutants on the occurrence and severity of chronic childhood wheezing illness in Nottingham school children

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June 1998–November 1999; Second Joint Research Programme

Introduction

Much interest has focused recently on the role played by outdoor air pollution in the aetiology of childhood asthma. However, exposure to pollution in the indoor environment is also likely to be an important determinant of personal pollution exposure. While it is unclear which, if any, of the many pollutants found in the home are playing a part in asthma aetiology, volatile organic compounds (VOCs), nitrogen dioxide (NO_2), environmental tobacco smoke (ETS) and damp have been identified as the most likely candidates based on evidence to date.

Volatile organic compounds originate from sources in the home such as furnishings, paints and cleaning agents, and several VOCs have been seen to have irritant effects on the airways at high doses. It is less clear what effect low-level exposure has on respiratory health, although some studies have reported an increased risk of asthma in relation to VOCs in the home (Norback *et al.,* 1993, 1995; Wieslander *et al.,* 1997). Many studies have investigated the respiratory effects of exposure to NO_2, and while there does not appear to be a clinically relevant effect on lung function, it remains unclear whether there is a relationship between NO_2 levels and the frequency or severity of asthma symptoms in children, and if so, at what threshold level. Several studies have suggested that maternal smoking is associated with an increased risk of respiratory symptoms in children of school age (Ware *et al.,* 1984; Cunningham *et al.,* 1996), but the impact of ETS exposure on the occurrence of allergic and non-allergic wheezing illness in school-age children has yet to be fully described. Epidemiological associations between damp housing and wheezing have been reported in children (Brunekreef *et al.,* 1989; Verhoeff *et al.,* 1995), possibly due to increased exposure to mould or house dust mite, although support for this hypothesis is limited.

This study will investigate the role of indoor pollution on the respiratory health of 9–10 year old children as an extension to the Nottingham Schools Study carried out in 1995, which assessed the aetiological effects of outdoor pollution from traffic and early life exposures on wheezing illness in schoolchildren. We will determine whether, and at what threshold level, exposure to each of a group of six VOCs, NO_2, ETS and dampness is associated with an increased risk of wheeze in children, controlling for various potential confounders. We will also establish whether exposure level is an independent determinant of the severity of asthma symptoms, peak flow variability, airway function or allergen skin sensitisation in the symptomatic children, and assess the modifying effect of specific allergen sensitisation.

Methods

Sample selection

In 1995 we surveyed 23 000 primary-school children in the Nottingham area, and obtained detailed aetiological data on 450 children in Year 1 (age 5–6) with parentally reported wheezing in the past 12 months. We also obtained detailed data on a one in four random subsample of 734 children in Year 1 and have now randomly selected 460 children from this group to provide us with a representative cross-sectional sample of this age group, which will also act as the source of our controls. Parents of the two groups of children were sent a letter describing the current study and asking about current wheezing status. Following the return of these letters we identified our case group as those with current wheeze in both 1995 and 1998, and our control group as non-wheezing children in both years. We are now contacting parents of cases, controls and those in the cross-sectional sample to arrange a time to visit the home.

Data collection and pollution monitoring

At the time of writing, 209 parents have agreed to take part and 130 children have been visited. During each visit, spirometry is performed to obtain measurements of forced expiratory volume in 1 s (FEV_1) and forced vital capacity (FVC), and skin prick tests to common allergens carried out to measure skin sensitivity to allergens. A saliva sample is taken for cotinine analysis to provide us with a measure of exposure to ETS. Cases are provided with a Peak Flow meter, and diary card to record peak expiratory flow (PEF) and symptoms twice daily for 4 weeks. In the kitchen, living room and child's bedroom an outside wall is probed with a Protimeter Surveymaster SM to measure the moisture content on or below the wall surface, and a visual assessment of mould growth made. A passive diffusion sampling tube used to measure NO_2 is left in the kitchen for 4 weeks and then sent to AEA Technology for analysis. In the child's bedroom, a Perkin-Elmer tube to measure VOCs is exposed for 4 weeks, and a sampling badge to measure formaldehyde levels for 3 days. These are sent to the Building Research Establishment (BRE) for analysis. During the visit we are also administering a questionnaire to the parents of all children to elicit details of gas appliances in the home, ventilation, recent painting or decorating etc., in collaboration with Derrick Crump at BRE. We aim to visit approximately 530 homes, comprising approximately 250 cases, 250 controls and 320 in the cross-sectional group, by March 1999. The study is due to be completed by the end of 1999.

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5.1.2 Exposure to indoor air pollution and the risk of upper and lower respiratory tract disease in asthmatic children and their mothers

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March 1998–February 2000; Second Joint Research Programme

Introduction

Nitrogen dioxide, particulates and ETS are the major indoor and outdoor pollutants that have been linked with respiratory symptoms in children and adults, particularly young women who cook with gas stoves. So far very few studies have focused on the indoor pollutants and their effects on asthma. Furthermore, many previous studies have not taken account of other indoor pollutants such as VOCs, carbon monoxide (CO) and confounding variables such as dampness and humidity. By measuring kitchen exposures to NO_2 (continuous and during cooking), particulates and ETS, CO and VOCs, and measuring levels of indoor allergens, dampness and humidity, we propose to show whether exposure to these factors increases the risk of respiratory episodes in asthmatic children and their mothers.

Phase I (pilot study)

Phase I of the study started on 16 March 1998 and finished on 6 April 1998. Patients were recruited from the asthma clinic of our local general hospital.

The pollutants measured and methods used were as follows.

- Nitrogen dioxide, both kitchen and personal exposures, was measured using passive sampling tubes.
- Carbon monoxide was measured using an electrochemical cell device manufactured by the Medical Engineering & Physics Department, Southampton University. Samples were taken at 20 s intervals for a period of 24 hours, the hourly averages were calculated on a sample-by-sample basis and then the maximum 1 hourly average of CO was identified.

Results

Results of the measurements of NO_2 and CO are summarised in Table 5.1.2a. The child personal NO_2 averaged over 7 days ranged from 2–40 ppb, median 11 ppb and the mother personal NO_2 averaged over 7 days ranged from 3–38 ppb, median 11 ppb. The continuous and during cooking NO_2 ranged from 4–45 ppb and 16–322 ppb, respectively, with medians of 9 and 129 ppb, respectively. The CO level ranged from 1–35 ppm, with a median of 3 ppm.

Table 5.1.2a Personal (child and mother) and kitchen (continuous and cooking) NO_2 and CO in the homes of nine families

Family number	Personal NO_2 (ppb)		Kitchen NO_2 (ppb)		Max CO (ppm)
	Child	Mother	Mean	Peak	
P001	2	3	5	16	2
P002*	11	16	37	154	4
P003*	12	10	6	129	2
P004*	33	38	45	253	13
P005	3	3	4	21	1
P006	10	8	9	26	1
P007*	40	20	24	140	35
P008	6	7	7	31	3
P009*	24	30	36	322	4

* Using gas cooking

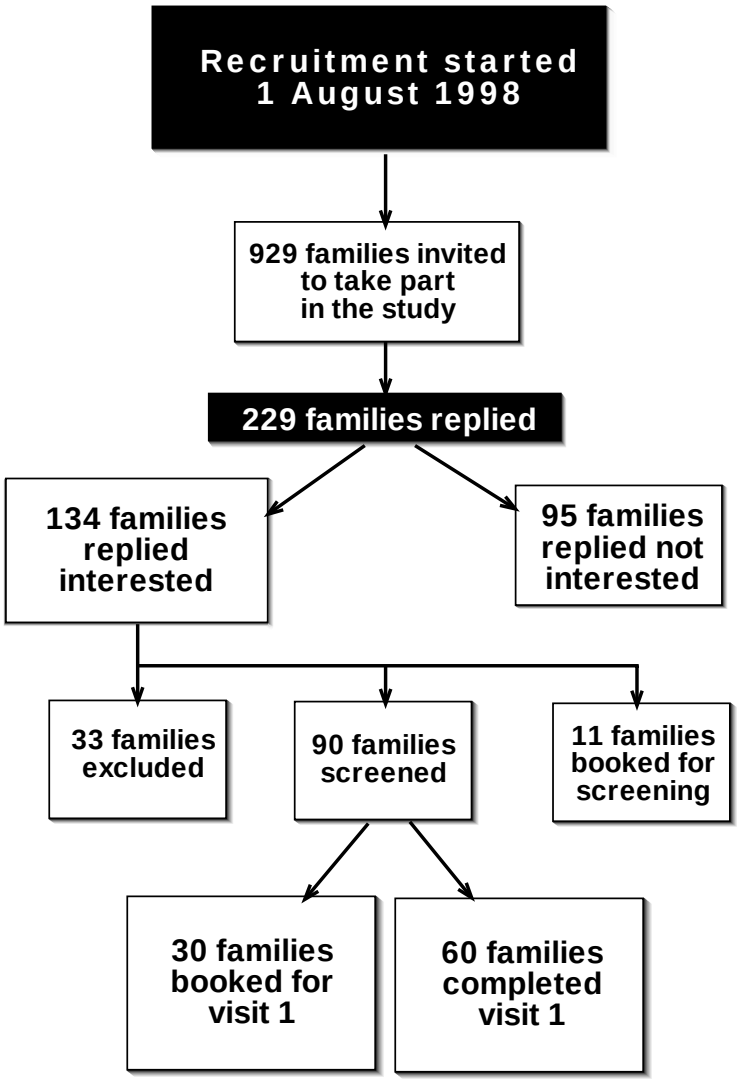
Phase II

The objectives of the longitudinal study are to:

- test whether the incidence, severity and duration of upper and lower respiratory tract symptoms are related to the exposure to indoor pollutants; and
- identify the important factors determining exposures to indoor pollutants.

The pollutants, allergens and variable confounding factors measured are: NO_2, CO, particulate matter <10 µm aerodynamic diameter (PM_{10}), VOCs, formaldehyde, ETS (urine cotinine), *Dermatophagoides pteronyssinus* allergen 1 (Der p1), dampness and humidity. The recruitment for, and progress of, the longitudinal phase of the study up to 25 December 1998 is illustrated in Figure 5.1.2a.

Figure 5.1.2a Recruitment and progress of the longitudinal phase of the study of indoor air pollution and asthma



5.1.3 Does indoor air pollution increase frequency, duration and severity of respiratory symptoms in an adolescent population?*

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February 1996–July 1997; First Joint Research Programme

Introduction

There is increasing public and scientific interest in the importance of indoor air pollution, particularly in the effects of NO_2, which, at levels encountered inside homes, could aggravate respiratory symptoms in susceptible individuals. Indoor pollution is, however, a complex aggregate of different pollutants and their additional effects are unknown.

In 1987 and 1995 a respiratory questionnaire was sent to 3033 children born between 1978 and 1980. The association between both persistent and late-onset respiratory symptoms and potential risk factors has been described elsewhere. A further study was designed to test the hypothesis that exposure to high levels of indoor air pollutants is associated with increased frequency, duration and severity of respiratory symptoms in this population now aged 16–18 years.

Questionnaire study

A detailed questionnaire was sent to all respondents of the 1995 questionnaire looking into the exposure to indoor air pollutants in 1987 and 1996. Out of the 2289 adolescents contacted, 1869 (79%) responded (M:F 1.13:1). Gas was used for cooking in 63% of households and for heating in 84%. A microwave was also used by 88% of the families. Univariate and multivariate logistic regression analyses were performed using the SPSS statistical package. Gas cooking, presence of gas central heating in the bedroom, and going to school by bus were negatively correlated with current wheeze while use of a microwave oven had an odds ratio of 1.6. Gas cooking and using a gas fire were negatively correlated with persistence of wheeze and going to school by bus was negatively correlated with late-onset wheeze. Multiple logistic regression analysis allowing for potential confounding variables was also performed and the only association that remained significant was between use of a microwave oven and current wheeze (OR 1.73, 95% CI 1.063–2.80).

Longitudinal study

Of the subjects symptomatic in 1995, 76 agreed to participate in the second part of the study, a detailed prospective panel study including the following: skin prick test, spirometry, urinary cotinine, indoor and personal NO_2 monitoring, VOC measurements, total suspended particulate (TSP) level, temperature, humidity, and measurement of Der p1 in household dust samples (lounge and bedroom). Measurements were performed in intervals at baseline, 3 months and 6 months. In addition, the subjects recorded their upper and lower respiratory tract symptoms (cold and chest score), morning and evening peak flow, the use of medication, the need to see a doctor, if they had to miss college or work, dust or smoke exposure at work or college and acute exposure to pollutants at home. The families also recorded the weekly gas and electricity meter readings.

Of the 76 patients, 38 (50%) were males, 61 (80%) were atopic (weal ≥3 mm for at least one of the aeroallergens), 51 reacted to house dust mite (83% of the atopics), and 48 (79%) to mixed grass pollen. At inclusion, mean FEV_1 was 89% (median 91%, range 54–119%) and at the last visit mean FEV_1 was 96% (median 96%, range 58–128%). The 7 month follow-up was completed by 69 patients (90%).

Results

Tables 5.1.3a and Table 5.1.3b show static levels of and personal exposure to a number of indoor pollutants. Table 5.1.3b shows measurements of NO_2 made during the study. Both personal and kitchen NO_2 levels were greater in households using gas for cooking compared with electricity (Mann-Whitney, $p < 0.01$) and with

* Completed by April 1998, abstract presented at second annual review meeting

unvented compared with vented or no gas appliances (Kruskal-Wallis, $p < 0.05$). However, there was no association between passive smoking or active smoking and NO_2 or total suspended particle levels (Tables 5.1.3a and 5.1.3b).

Conclusion

It has not been possible to show any association between gas cooking and respiratory symptoms in this adolescent population. The only significant association after controlling for all potential confounding variables is between use of a microwave oven and current wheeze. Recent studies have suggested some deleterious effects from microwaves and this should be further investigated since we have shown that a large proportion of the population is now using this method of cooking.

In homes where gas was used for cooking, levels of NO_2 were within the range expected from previous studies and at levels that are thought to be deleterious to health. Use of gas for cooking in the home increased exposure to NO_2 even in an age group thought previously to be less affected by domestic levels because they spend more time outside the home.

Total suspended particles were recorded over 24 hours and during this time all homes demonstrated at least one peak at a very high level (range 330–2550 mg/m^3) although mean values in all homes were below the 24 hour US ambient air quality standard of 260 mg/m^3. Strikingly, some of these peaks were found during night-time hours when there was no activity inside the house and these peaks might be a reflection of outdoor levels. To date, no study has investigated the health outcome of these very short but intense peaks.

Table 5.1.3a Static and personal measurements of selected indoor air pollutants (mg/m^3)

Pollutant	Mean	Median	Interquartile range
NO_2			
Personal	19.8	16.7	12.1–26.1
Static	44.5	32.5	20.7–56.0
VOCs			
Benzene	7.3	4.8	4.0–7.7
Toluene	77.8	70.0	43.6–98.1
Xylene	40.4	32.1	23.0–43.8
Decane	11.8	9.3	5.4–16.0
Total (17 compounds)	354.3	332.3	210.6–438.4
TSP			
24 hour mean	66.2	58.5	43.0–80.0
10 s peak	1604	1550	915–2357

Table 5.1.3b Use of gas appliances and NO_2 levels (mg/m^3)

Sample	Mean	Median	Interquartile range
Personal			
No gas used	15.2	12.7	10.2–19.0
Vented gas appliances	20.4	19.5	15.3–21.4
Unvented gas appliances	26.5	24.3	17.0–32.6
Kitchen (static)			
No gas used	26.4	21.7	15.1–28.8
Vented gas appliances	46.2	38.7	31.0–58.3
Unvented gas appliances	70.7	65.0	38.5–102.6

5.1.4 Carbon monoxide poisoning in a smoker*

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Introduction

Acute exposure to high levels of CO is associated with morbidity and mortality (Ernst & Zibrak, 1998). The symptoms of acute CO poisoning include headache and malaise, ischaemic cardiac pain and neurological abnormalities of all grades leading to convulsions, coma and ultimately death. A proportion of patients will develop a late neuropsychiatric syndrome. The correlation between circulating carboxyhaemoglobin (COHb) levels, neurological impairment and prognosis in acute poisoning is poor (Hardy & Thom, 1994). The situation in relation to lower level chronic CO exposure is less clear. Again symptoms such as headache, nausea, malaise and gastrointestinal upset have been attributed to chronic low-level exposure from a variety of sources. However, cigarette smokers commonly have COHb levels of the order of 5–7%, yet do not in general describe associated symptoms. This report concerns an individual with CO exposure apparently attributable to cigarette smoke alone, in whom the level of exposure was sufficient to cause symptoms.

Case report

A 28 year old male insulin-dependent diabetic reported a 10 week history of headache and general malaise. On one occasion, he had lost consciousness while at home. He had been given intramuscular glucagon by a family member on the assumption that this was a hypoglycaemic event, but had no response. However, he gradually regained consciousness over a period of 2 hours. Medical help was not sought. Subsequent measurements of COHb were made by his GP on four occasions over a 1 month period: values of 12%, 14%, 15% and 17% were obtained. His house and works van were screened for CO with negative results. He was employed outdoors as a fence builder and admitted to smoking 40 cigarettes per day. At review in the respiratory clinic, he was normotensive with normal cardiovascular and respiratory examination. He was slow at performing serial sevens but was otherwise neurologically intact. Exhaled CO was elevated at 51 ppm (reference level for nonsmoker, 3 ppm). He was admitted to hospital for observation over 24 hours and reduced his cigarette consumption to 15 during this period. His COHb level at the end of this period was 7%. He was then given a hyperbaric oxygen treatment at 200 kPa oxygen for 90 min. Immediately following this his COHb level was 1%. After a 4 hour post-treatment period in which he was deprived of cigarettes, his COHb level remained static at 1.2%. During this period he reported that his headache and malaise had completely resolved. He was strongly advised to stop smoking. On review 3 weeks later, he had unfortunately returned to his previous smoking habit, his symptoms had returned and his COHb level had risen to 14%.

Discussion

The chronic symptomatology in this patient, his episode of collapse with subsequent slow spontaneous recovery, and the response of his chronic symptoms to treatment was compatible with CO poisoning. His symptoms were associated with measured COHb levels and the only identifiable source of exposure was his cigarette habit. Screening of his environment was otherwise negative, he was employed outdoors and he had no significant exposure to xenobiotics, which can induce endogenous CO production. The latter possibility was also made unlikely by the failure to reaccumulate COHb when he was deprived of cigarettes. It has previously been suggested that symptoms of chronic CO poisoning occur only at COHb levels above 10% (Heckerling *et al.,* 1988). Our findings in this patient would be compatible with that observation. The development of symptoms in chronically exposed patients may occur only if the exposure is sufficient to raise COHb levels above those usually seen in smokers, or if there is intermittent high level exposure on a background of lower level exposure. The important contribution of smoking to chronic CO poisoning must be borne in mind. There is a need for further studies of the pattern of exposure in subjects complaining of symptoms that might be attributable to chronic CO poisoning.

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5.2 Damp housing and health

5.2.1 An epidemiological study of the impact of damp and other aspects of poor housing on adult health

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April 1998–March 1999; Second Joint Research Programme

Introduction

It is well known that the relationship between damp housing and health is complicated. Many factors associated with housing dampness, such as social class, occupation, and lifestyle, also affect health (Keithley *et al.,* 1984). This study aimed to contribute to the understanding of damp housing-health relationships by examining data on housing, health, and a wide range of contextual variables from a large cross-sectional study, the third Oxford Healthy Life Survey (HLS III).

Methods

The HLS III questionnaire was administered, by post, in 1997, to a representative sample of 14 868 adults aged 18 to 64 years living in Oxfordshire, Buckinghamshire, Berkshire and Northamptonshire. Data from the HLS III are useful in examining associations between housing and health for two reasons. First, the data include information on three health outcomes: self-reported long-standing illness including asthma; the anglicised version of the Medical Outcomes Trust Short Form 36 (SF-36); and aspects of health service use. Second, they include information on reported housing dampness (three categories: 'no problem'; 'more of a nuisance than a problem'; 'a serious problem') and a broad range of sociodemographic, behavioural and environmental variables (see Table 5.2.1a), allowing an examination of health–housing associations taking into account other factors.

Relationships between damp housing and health were initially examined using the chi-squared test and one-way analysis of variance. Associations demonstrated were then further examined in the context of other independent variables using logistic and linear regression. For the purposes of all these analyses, a 'strong association' is considered to exist where $p < 0.01$.

Results

A response rate of 64% (8889)[b] was achieved for the HLS III. A comparison with the 1991 census showed that respondents were broadly representative of the local

[b] 1068 deletions were made because of, e.g., 'unknown addressees'

Table 5.2.1a Variables used in multivariate modelling

Sociodemographic	Lifestyle	Housing environment
Age	Smoking	Damp housing
Social class[a]	Passive smoking	Housing tenure
Marital status	Alcohol consumption	Cold housing
Employment status	Physical exercise	Overcrowding
Age left full-time education	Fruit & vegetables	Burglary
Ethnicity		Vandalism
Worry about money		Assaults and muggings
Worry about job security		Exposure to speeding traffic
Worry about pressure at work		Discarded needles and syringes
		Nuisance from dogs
		Smells and fumes
		Noise
		Litter and rubbish
		Disturbance by neighbours
		Lack of open spaces
		Poor street lighting
		Poor public transport
		Disturbance by children or youngsters
		Uneven or dangerous pavements
		Desire to move away

[a] Social class based on own occupation was coded according to the Registrar General's classification

population in terms of age, sex, social class and ethnicity.

Reported dampness was found to be associated with self-reported asthma (chi-squared test, $p < 0.001$), greater use of doctors' surgeries, casualty and outpatients' departments (chi-squared test, $p \leq 0.001$) and poorer scores for all eight dimensions of the SF-36 (one-way ANOVA, $p < 0.001$). A dose–response relationship was demonstrated for all these associations.

The association between reported asthma and reported housing dampness was further explored by logistic regression, adjusting for the variables listed in Table 5.2.1a. Being unable to keep the home warm enough in winter was the only housing variable strongly associated with asthma ($p < 0.01$). This variable was also associated with frequent (four or more) visits to the GP and visits to hospital outpatients' departments ($p < 0.01$). A number of other sociodemographic and lifestyle variables were included in these final models, most commonly age, sex and employment status. Reported dampness did not feature in any of these models.

The associations between health status, as measured by the eight dimensions of the SF-36, and reported housing dampness were further explored using linear regression, again adjusting for the range of variables in Table 5.2.1a. The variables in the final models depended on the dimension of the SF-36 being examined. Once again, the most important housing variable was being unable to keep the home warm enough in winter. This variable was associated with poorer scores for all eight dimensions of the SF-36 ($p < 0.001$). The next most important housing variable, 'a lack of open spaces', was strongly associated ($p < 0.01$) with seven dimensions. Other variables associated with all eight dimensions were: employment status, vigorous exercise, worry about money and worry about pressure at work ($p < 0.01$). Damp housing was only strongly associated ($p < 0.01$) with 'health perception', and weakly associated ($0.05 > p > 0.01$) with 'physical function', 'role limitation-mental' and 'role limitation-physical'.

Discussion

It is clear that, for this data set, the importance of reported damp housing as a factor in reported ill-health, seen in the preliminary analyses, is diminished when other variables are taken into account in the statistical modelling. The most important housing variable was the perceived temperature of the home, not reported dampness.

It is important to consider why reported dampness does not appear to be a significant factor in these analyses, as it has been consistently reported in the literature. Damp housing is directly related to cold housing as relative humidity declines with increasing temperature. Univariate analyses indicated a linear association between reported damp housing and being unable to keep the home warm enough in winter (chi-squared test, $p < 0.001$). It is therefore possible that reported cold housing reflects the combined effects of reduced temperature and increased humidity on health. However, it is clear that, when both damp and cold housing are entered into the multivariate models, it is the cold rather than the damp variable that predominates. It is important to note that, despite a considerable literature on housing dampness, cold housing has been somewhat neglected and rarely examined independently of dampness. The only study that appears to have modelled the effects of cold and dampness together is Platt *et al.* (1989). Here, perceived cold, along with other housing factors, was considered in an examination of housing dampness and health. The association between damp/mouldy housing and number of symptoms persisted when the other housing factors were taken into account.

It is possible that cold housing is affecting health in its own right. The dangers of very low temperatures are well established (Lowry, 1991), but there appears to be little information on less extreme temperatures. Lesser degrees of cold in low indoor temperatures may damage the lungs indirectly by reducing the resistance of the body to infections secondary to colds and influenza (Collins, 1986).

Conclusions

This study indicates that being unable to keep the home warm enough in winter is more strongly associated with health outcomes than reported dampness in the home. However, as reported cold housing and reported dampness in the home are so closely related, this does not mean that damp in the home is unimportant. It is likely that the combined effects of cold, damp housing are having an effect. The apparent importance of reported cold over reported damp housing in this survey does, nevertheless, merit some further consideration.

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5.2.2 Consequences for health and effects of damp on the incidence of airborne microbial cell wall components in the home*

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February 1998–July 2000; Second Joint Research Programme

This project will address the following questions.

- Does a damp home play a role in triggering asthma and, if so, by what mechanisms?
- Does a damp home contribute to the prevalence and incidence of other respiratory illnesses?
- Are those living in a damp home more sensitive to the effects of other indoor air pollutants or allergens?

A case–control study will incorporate environmental measurements (personal exposure monitoring and subsequent modelling), examine blood indicators and measure lung function.

Up to 120 persons (about 50 to 60 homes) will be recruited via the local press in approximately equal numbers from Luton and Sheffield. Each home will be surveyed and classified according to dampness and severity of visible mould growth. Temperature and relative humidity will be measured using non-intrusive micro-dataloggers.

Exposure to total (viable and non-viable) bacteria and fungi, endotoxin and (1→3)-β-D-glucan in airborne and settled dust will be quantified. Total inhalable dust will be quantified to examine the relationship between dustiness and microbial contamination. Air monitoring using filtration samplers (e.g. 37 mm Millipore cassettes and IOMs) twice per annum and a limited amount of general area monitoring using six-stage Andersen samplers will be undertaken. Settled dust from carpets, soft furnishings, shelves and light fittings will be collected onto paper filters using vacuums fitted with ALK allergen mouthpieces.

Exposure within the home and the potential causal relationships between damp, bacterial/fungal growth, microbial cell wall components and illness will be determined from the results of questionnaires and lung function and blood tests on residents.

- Questionnaires will establish reported respiratory symptoms and potential influential factors, e.g. work history, smoking.
- Lung function measurements will include FEV_1 and FVC. Methacholine challenges will be undertaken. Peak flow variability in excess of 20%, in conjunction with a symptoms diary, will be monitored.
- Blood will be taken and measured for serum markers of airway inflammation, atopic status and allergic sensitisation to prevalent fungi. Flow cytometry will be used to investigate changes in immune cell responses, e.g. activated helper T-cells ($CD3^+$ $CD4^+$ $CD25^+$). Expression of CD14 on peripheral blood monocytes will also be measured as a marker of endotoxin exposure.

These data will improve understanding of the role that damp might play in determining the amount of airborne endotoxins and glucans in indoor environments. They will also be used to assess the role of microbial cell wall components in respiratory ill health and airway inflammation attributable to damp homes to determine risk to exposed groups. Risk factors will be modelled in terms of exposure, potential mitigating circumstances and risk minimisation, e.g. use of ventilation and type of heating.

* Abstract presented at second annual review meeting

5.3 Airborne particles and health

5.3.1 Risk factors for acute respiratory events in the Thamesmead Respiratory Health Survey

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January 1998–October 1999; First Joint Research Programme

Introduction

Patients with pre-existing respiratory disease may be at risk of acute respiratory symptoms on days when air quality is poor. Also, patients with asthma may be more sensitive to airborne allergens that may be present on days when concentrations of air pollutants, especially PM_{10}, are elevated. The aim of this study is to assess risk factors for acute respiratory symptoms in patients who have asthma and/or chronic obstructive pulmonary disease (COPD), and to estimate the association between symptom exacerbation and air pollutant levels during the study period. The study is also collecting particulate pollution in order to determine whether patients experiencing symptoms are more likely to have specific IgE antibodies directed to components of PM_{10} than are symptom-free patients.

The study is based in a large general practice in south-east London that has a patient population (aged 18–64) of approximately 11 000, of which approximately 850 patients have a diagnosis of asthma and/or COPD. The study has two phases: a cross-sectional survey of patients who have been prescribed at least one bronchodilator in the last 12 months; and a longitudinal phase in which patients recruited into the study are monitoring their respiratory symptoms for 12 months.

Data collected

A total of 827 eligible patients were identified and 296/827 (35.8%) patients were recruited into the study, 271 (32.8%) refused and 259 (31.3%) did not respond to our letters. Examination of a randomly selected sample of 20% of the non-responders indicated that 25% of this sample are no longer registered with the practice.

Patients were seen in the health centre between April 1997 and July 1998 by the study nurse, and information was collected on baseline and post-bronchodilator lung function (FEV_1 and FVC), atopic status (determined by skin prick test to common allergens) and plasma antioxidant status (vitamins A, C, E and uric acid), as well as questionnaire data on respiratory symptoms, occupation, use of medication, diet and smoking habits.

The symptoms monitoring phase of the study is continuing until July 1999. The patients report acute respiratory symptoms to the study by telephoning a freephone number. Information on respiratory symptoms reported to the GP is obtained from the patients' computer notes.

Results

Table 5.3.1a shows the number of patients who have reported acute respiratory symptoms events, either to the study or to their GP, from the time they were recruited into the study to July 1998.

Sixty-five patients reported a total of 102 acute respiratory symptoms events to the study. Twenty-two of these patients also reported 41 separate events to their GP and a total of 75 other patients who did not report any events to the study reported 141 events to their GP.

Table 5.3.1b shows the results of lung function tests in patients who reported any acute respiratory symptom either to the study or to the GP.

There was no relationship between plasma antioxidant levels and reporting an event. Patients who reported ever having had asthma or having had an attack of asthma in the last 12 months were not at increased risk of reporting events when compared with other patients

Table 5.3.1a Number of patients reporting events to the study and/or to the GP

Patients reporting to the study	Patients reporting to the GP			Total
	No	Yes, any respiratory symptom	'Asthma' recorded in notes	
No	156	75	35	231
Yes	43	22	12	65
Total	199	97	47	296

Table 5.3.1b Summary of lung function tests in patients with and without reported events

Lung function measure	Mean (95% CI)		Difference (95% CI) value
	In patients not reporting events	In patients reporting events	
FEV_1	2.88 (2.73, 3.02)	2.47 (2.33, 2.61)	-0.41 (-0.20, -0.61) p <0.001
FVC	3.60 (3.42, 3.78)	3.20 (3.04, 3.36)	-0.40 (-0.15, -0.64) p = 0.007
$pbFEV_1$	3.01 (2.85, 3.16)	2.58 (2.44, 2.73)	-0.42 (-0.21, -0.64) p <0.001
$impFEV_1$	3.29 (2.21, 4.36)	4.47 (3.20, 5.74)	1.18 (-0.51, 2.86) p = 0.915
FEV_1diff	0.11 (0.08, 0.13)	0.10 (0.08, 0.13)	-0.01 (-0.04, 0.04) p = 0.474

$pbFEV_1$, post-bronchodilator FEV_1 (FEV_1 measured 10 min after two inhalations of 100 mg each of salbutamol from an MDI via a volumatic spacer); $impFEV_1$, per cent improvement in FEV_1 following bronchodilator challenge; FEV_1diff, increase in FEV_1 following bronchodilator challenge

with COPD in the study (Odds ratio (OR) for asthma ever = 1.01, 95% CI 0.38–2.70; OR for asthma in last 12 months = 1.67, 95% CI 0.90–3.09). Smoking was not an additional risk factor for respiratory exacerbations in this group of patients (OR = 1.27, 95% CI 0.98–1.64); however, older patients were more likely to report acute respiratory symptoms (OR for a 10 year increase in age = 1.21, 95% CI 1.01–1.45).

Although atopy (positive skin prick test to any allergen) was not a risk factor for respiratory exacerbations (OR = 0.76, 95% CI 0.48–1.20), when sensitisation to individual allergens was examined it was found that patients who were sensitised to grass were less likely to report symptoms (OR = 0.58, 95% CI 0.35–0.95).

Conclusions and further work

Preliminary analyses suggest that poor initial lung function is a risk factor for acute respiratory exacerbations. The patients who have reported events to the study are not more likely to be atopic, or have a diagnosis of asthma but they are likely to be older and have FEV_1 that does not improve following bronchodilator challenge, suggesting that those with fixed airway obstruction have a greater risk of subsequent exacerbations. The relation of exacerbations to different forms of air pollution and how this might vary between subgroups of patients has not yet been assessed.

As the symptoms monitoring phase is still under way we have yet to carry out a nested case–control analysis of reported respiratory symptoms and to assess the relation between symptoms and air pollution levels. In addition the last phase of the study will include laboratory investigations to determine whether patients with symptoms have specific IgE antibodies to extracts of PM_{10} collected on the day(s) when they had their symptoms. As indoor allergen exposure can be an important trigger for exacerbations of symptoms, all patients were invited to have a home visit to collect samples of dust for indoor allergen measurement. These are currently being assayed for Der p1 (house dust mite allergen), *Felis domesticus* allergen 1 (Fel d1) cat allergen and *Blattella germanica* allergen 2 (Bla g2) cockroach allergen.

5.3.2 Acute health effects of ultrafine particulate air pollution

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March 1996–March 1999; First Joint Research Programme

Introduction

This study is designed to test the hypothesis that measurements of the number of ultrafine particles better predict health effects than do measurements of PM_{10}. In a pilot study during the 1997/8 winter, of only 15 subjects, a significant relationship was found between cumulative 3-day means of ultrafine particle counts and mean scores for shortness of breath ($p = 0.001$; Figure 5.3.2a). Meanwhile, there was no such relationship with PM_{10} ($p = 0.966$; Figure 5.3.2b). However, when the relationship between ultrafine particle counts and shortness of breath was corrected for autocorrelation and temperature, the statistical significance dropped (to $p = 0.08$). The hypothesis is now being retested in a larger population.

The aim of the study is to measure the coarse, fine and ultrafine fractions of ambient particles in Aberdeen and relate these data to the health of a group of patients with chronic lung disease. These subjects are making subjective and objective assessments of their chest condition. Symptom scores, use of extra doses of bronchodilators and peak expiratory flow measurements are being recorded in a diary for 3 months during the winter of 1998/99. These health indices will then be correlated with numeric and gravimetric measurements of ambient 24 hour daily concentrations of coarse and fine particles measured as PM_{10} and $PM_{2.5}$, respectively, and counts for ultrafine particles (<100 nm).

The objectives are to determine:

- the daily variation in symptoms, peak flow rates and bronchodilator use in a group of elderly subjects over 3 months;
- the daily variation of ambient PM_{10}, $PM_{2.5}$ and ultrafine particles in Aberdeen; and
- the daily variation in mean ambient temperature, pressure, humidity and wind speed.

Methods

Air pollution data

The weather is thought to be a possible confounder in air pollution studies. Hence, continuous hourly measurements of daily mean values of ambient temperature, pressure, humidity and wind speed are being made by the Skyeminimet SDL 2990.

PM_{10} and $PM_{2.5}$

PM_{10} measurements will be obtained from the Rupprecht and Patachnick, Tapered Element Oscillating Microbalance (TEOM) employed by Aberdeen District Council's Environmental Section. Data from this instrument will be converted into daily means. $PM_{2.5}$ measurements are being made by the TSI Dusttrack sampling continuously and recording averages every 30 min.

Ultrafine particles

Counts of ultrafine particles are being made by the TSI Scanning Mobility Particle Sizer (SMPS). The SMPS system comprises the combination of a Model 3071A Electrostatic Classifier and Model 3022A Condensation Particle Counter (CPC). This system is programmed to make hourly measurements in two scans of particles between 10 and 500 nm in size.

Subjects

Forty-four subjects aged over 50 years, of both sexes, with either asthma or COPD were recruited from the chest clinics at the Aberdeen Royal Infirmary. They all have clinically significant disease requiring treatment and claim to notice a change in their symptoms with changes in the weather. Informed consent has been obtained and letters of notification have been sent to their general practitioners.

Each subject is keeping a daily diary of symptom scores, drug use and peak flow measurements for 3 months through the 1998/99 winter. The first set of three lots of

Figure 5.3.2a Relationship between ultrafine particle counts and shortness of breath

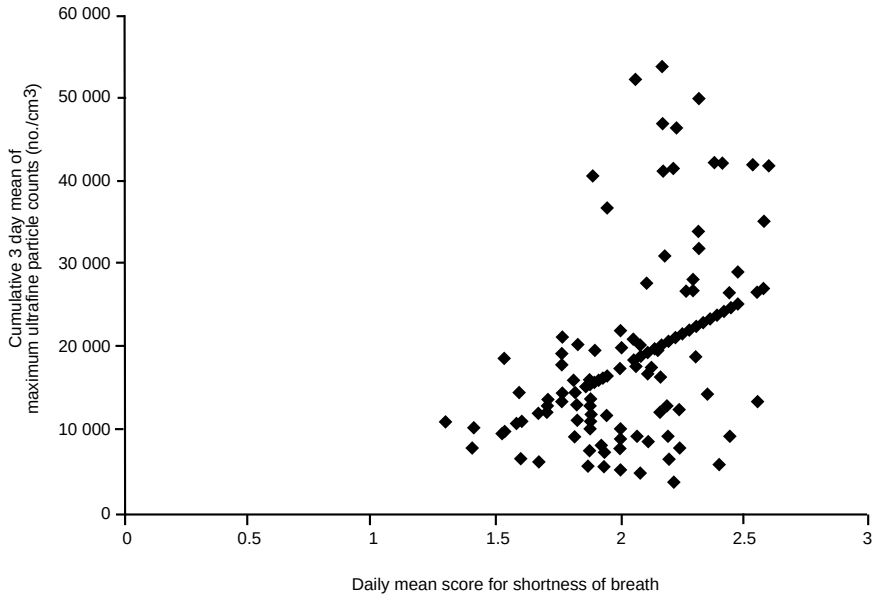
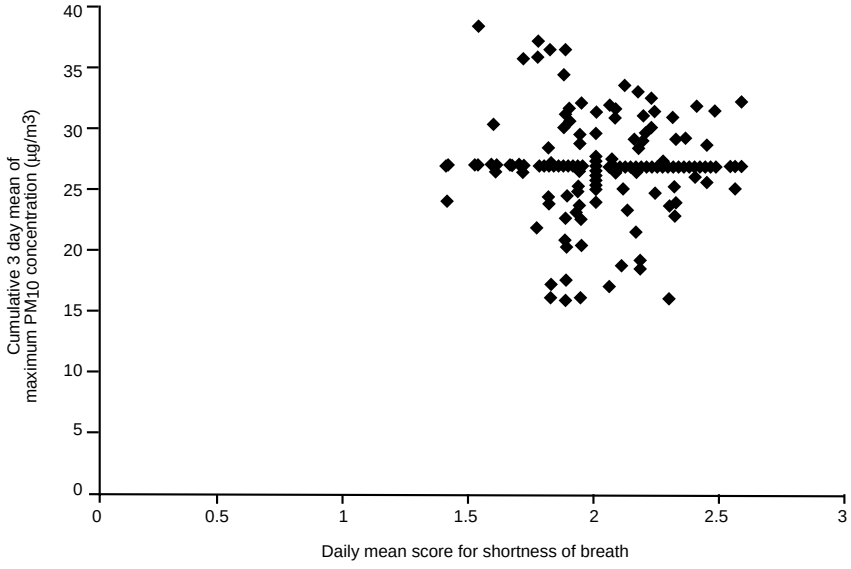


Figure 5.3.2b Relationship between PM_{10} and shortness of breath



diaries was expected back by the first week of 1999. The second set were to be sent to them by post before completion of their current diaries.

Data analysis

All data will be managed on a standard database. For each measured response, each subject will have a time-ordered series of response measurements. Responses such as peak expiratory flow rate will be measured on continuous scales while the other health indices will be regarded as categories, such that the binary presentation can be fitted into logistic regression models.

Duration

Actual data recording commenced on the 1 December 1998. The 3 month data collection was to have been complete by March 1999 at the latest.

5.3.3 Health effects of urban air pollution, and susceptibility to them: Further evidence from a time-series and a cohort study

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February 1996–February 1998; First Joint Research Programme

Introduction

We have previously described our findings showing a significant association between changes in urban 'black smoke' (BS) concentration and respiratory mortality, as well as between PM_{10} and cardiovascular morbidity (Prescott *et al.,* 1998). We present further analyses of factors that might indicate susceptibility to particulate air pollution in this time-series study, and further data from a cohort study.

Methods

Daily BS measurements at a monitoring station near the city centre were obtained from the Environmental Services Department of Edinburgh District Council, and PM_{10} data were obtained from the enhanced urban network. For the time-series study, data were obtained from the Scottish Record Linkage System on all emergency hospital admissions with a cardiorespiratory principal diagnosis for Edinburgh residents over the period 1 January 1981 to 30 June 1995. A patient identifier allowed such records to be linked, and to each record was appended the date and cause of death, if it occurred in the study period. In defining the issue of susceptibility in mortality, it was decided to focus on the mortality of people who had not lived for longer than 3 years after their last hospital admission. However, since recently discharged patients might behave in a different way, it was also decided to limit further the analysis to those whose hospitalisation as an emergency was more than 6 months prior to their death. Poisson regression modelling was used to relate the daily number of deaths to particulate pollution averaged over the previous 3 days, after controlling for month and minimum temperature and wind speed on the day of death (Prescott *et al.,* 1998). The cohort study was based on 1592 subjects aged 55–74 on recruitment between August 1987 and November 1988. All fatal and nonfatal myocardial infarctions (MIs) and strokes (collectively referred to as cardiovascular events) were included if they occurred between recruitment and 31 March 1998. Fibrinogen and whole blood and plasma viscosity were measured on recruitment and approximately 5 years later.

Results

In the study period, 1981 to mid-1995, there were 17 571 non-accidental deaths of Edinburgh residents who had had at least one previous cardiorespiratory emergency admission, and had died more than 3 days after their last such admission. Table 5.3.3a shows the estimated relative risks of death associated with a 10 $\mu g/m^3$ increment in black smoke averaged over the previous 3 days. For deaths from respiratory causes in the interval of 6 months and 3 years after their most recent cardiorespiratory emergency admission there was a statistically significant association, with an estimated 12% increase in risk.

The rheological and fibrinogen measurements showed strong associations with most of the classical risk factors for heart disease. Black smoke showed a negative relationship with fibrinogen at the first visit but a positive relationship at the second visit, while PM_{10} showed a weak positive relationship at the second visit. The change in fibrinogen between the first and second visit showed no significant correlations with changes in BS over the same period, but was significantly correlated with the difference in minimum temperature. The only meteorological variable to show any significant association with rheology was wind speed.

In the cohort study, 384 subjects died before 31 March 1998, thus leaving a total of 5 271 882 person days of exposure. During this exposure there were 343 cardiovascular events in 273 subjects and 117 of these events were fatal.

Modelling of the cohort data and of a nested case–control study within it, showed trends suggesting that individuals in the higher quintile of baseline

Table 5.3.3a Estimated relative risk of death associated with a 10 μg/m^3 increment in black smoke averaged over the previous 3 days

	Cardiovascular deaths					
	Following any cardiorespiratory admission			Following any cardiovascular admission		
Lag (days)	No. deaths	RR	95% CI	No. deaths	RR	95% CI
4–180	3507	0.99	0.94,1.03	2841	1.00	0.95, 1.05
181–1095	3190	1.03	0.98,1.08	2425	1.03	0.98, 1.09
>1095	2094	1.00	0.93,1.08	1606	1.03	0.95, 1.12)
	Respiratory deaths					
	Following any cardiorespiratory admission			Following any respiratory admission		
Lag (days)	No. deaths	RR	95% CI	No. deaths	RR	95% CI
4–180	797	0.96	0.87, 1.07	464	0.97	0.86, 1.10
181–1095	777	1.12	1.02, 1.21	330	1.14	0.99, 1.31
>1095	474	0.96	0.84, 1.10	159	–*	–*

* Estimates were not calculated for this model because there were too few deaths

fibrinogen might have a higher cardiovascular event rate for a given increment in particulate pollution.

Discussion and conclusions

Susceptibility to adverse health effects of air pollution may operate at several levels, ranging from inherited genetic factors, through to prior exposures, reduced functional reserve or overt disease. These have been summarised in Table 5.3.3b. In our studies we have so far presented data relating to categories 3 and 7, although work addressing other categories is in progress.

The central estimates in the time-series study are consistent with a threefold increase in susceptibility in people of the same age group in the previously hospitalised subset. This trend warrants further investigation with a more powerful study design, since it might help to define a susceptible subgroup of the population.

Small but significant effects of environmental factors on rheological measurements can be detected. However, they are not consistent nor as strong as those from classical risk factors for heart disease (such as sex, smoking, blood pressure or cholesterol). These classical factors should be included in any multiple regression analysis to examine the adjusted effects of pollution, mainly with the aim of improving power to detect relationships with pollution.

Table 5.3.3b Factors affecting susceptibility to adverse health effects of air pollution

Category	Examples
1. Genetic	Fibrinogen polymorphisms
2. Previous exposure	Tobacco smoking
3. Classical risk factors	Plasma fibrinogen, LDL-cholesterol
4. Pre-existing symptoms	Angina, chronic bronchitis
5. Impaired function	Reduced spirometric values
6. Confirmed disease	Ischaemic heart disease, bronchial asthma
7. Prior emergency admission	Myocardial infarction, COPD exacerbation
8. Other factors	Age, socioeconomic status

Further analysis taking account of confounding variables by means of a nested case–control approach is under way to explore possible main effects of pollution as well as susceptibility in certain cohort subsets.

There is need to investigate trends in our data suggesting that the presence of classical cardiovascular risk factors might be associated with an increased risk of ill health from subsequent exposure to particulate pollution.

References

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5.3.4 Air pollution and cardiovascular disease: An investigation of the relationship between particulate air pollution and blood coagulation factors

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Introduction

Particulate air pollution has been associated with excess deaths from, and increases in hospital admissions for, cardiovascular disease among older people. We have investigated whether this might be a consequence of alterations in the blood, caused by the action of fine particles on alveolar cells, by making repeated measurements of haematological factors in older people and relating these to measurements of airborne particles.

Methods

One hundred and twelve individuals aged 60 years or more in Edinburgh and Belfast provided repeated blood samples over 18 months. Estimates of individual exposure to PM_{10} were made for each 3 day period prior to blood sampling. The relationships between blood values and both personal exposure estimates and city centre measurements of PM_{10} were investigated by analysis of covariance, adjusting for city, season, temperature and repeated individual measurements.

Results

Estimated personal PM_{10} exposure over the 3 days prior to blood sampling showed strong negative correlations with haemoglobin concentration, packed cell volume (PCV) and red blood cell count ($p < 0.001$) and weaker negative correlations with platelets and factor VII ($p < 0.05$) (Table 5.3.4a). The changes in red cell indices persisted after adjustments had been made for plasma albumin in a sample of 60 of the subjects (Table 5.3.4a). City centre PM_{10} measurements over 3 days also showed negative correlations with haemoglobin and red cell count ($p < 0.001$) and, less strongly, with PCV and fibrinogen ($p < 0.05$), the relationship with haemoglobin persisting after adjustment for albumin (Tables 5.3.4a and 5.3.4b). C-reactive protein showed a positive association with city centre measurements of PM_{10} ($p < 0.01$). The estimated changes in haemoglobin associated with an alteration in particle concentration of 100 µg/m^3 were 0.44 g/dl (95% CI 0.62–0.26) for personal PM_{10} and 0.73 g/dl (1.11–0.36) for city centre PM_{10}.

Discussion

This investigation is the first to have demonstrated associations between haematological indices and air pollution. The changes in haemoglobin adjusted for albumin suggest sequestration of red cells in the circulation, and we propose that the action of fine particles on lung endothelial cells might be responsible for changing red cell adhesiveness. Peripheral sequestration of red cells offers an explanation of the observed cardiovascular effects of particulate air pollution.

Table 5.3.4a Results of mixed model analysis of covariance relating blood measurements to estimated individual exposures and city centre measurements for the previous 3 days, expressed as change per 100 μg/m^3 rise in mean daily PM_{10}

Blood variable	3 day personal exposure estimates			3 day city centre PM_{10}		
	Mean change	95% CI	p value	Mean change	95% CI	p value
Haemoglobin (g/dl)	-0.44	-0.62, -0.26	<0.001	-0.73	-1.11, -0.36	<0.001
Packed cell volume (ratio)	-0.016	-0.022, -0.01	<0.001	-0.013	-0.025, -0.001	0.029
Red cell count (x10^{12}/l)	-0.14	-0.20, -0.08	<0.001	-0.18	-0.29, -0.07	0.001
Platelets (x10^{12}/l)	-10.8	-21.2, -0.4	0.039	+9.8	-11.4, +31.0	0.36
White cell count (x10^9/l)*	-1%	-5%, +4%	0.67	-2%	-11%, +8%	0.61
Interleukin-6 (pg/ml)*	-7%	-28%, +21%	0.59	+10%	-37%, +91%	0.73
C-reactive protein (g/l)*	+7%	-36%, +36%	0.73	+147%	+20%, +477%	0.014
Fibrinogen (g/l)*	-1%	-6%, +4%	0.80	-9%	-19%, 0%	0.042
Factor VII (% standard)*	-7%	-14%, -1%	0.014	+1%	-11%, +15%	0.88

* These values have been log transformed and are expressed as mean percentage change per 100 μg/m^3 increase in PM_{10}

Table 5.3.4b Change in blood variables (with 95% CI) in relation to a 100 μg/m^3 increase in mean daily PM_{10} over 3 days in a sample of 60 subjects, before and after adjusting for plasma albumin

Blood variable	Direct association		Adjusted for albumin	
	Personal exposure	TEOM	Personal exposure	TEOM
Haemoglobin (g/dl)	-0.37 (-0.55, -0.18) p <0.001	-0.6 (-1.06, -0.13) p = 0.012	-0.30 (-0.47, -0.12) p <0.001	-0.46 (-0.90, -0.02) p = 0.039
Packed cell volume (ratio)	-0.013 (-0.019, -0.007) p <0.001	-0.008 (-0.024, +0.008) p = 0.30	-0.011 (-0.017, -0.005) p <0.001	-0.004 (-0.018, +0.010) p = 0.61
Red cell count (x10^{12}/l)	-0.10 (-0.17, -0.057) p <0.001	-0.133 (-0.28, +0.01) p = 0.07	-0.087 (-0.141, -0.033) p = 0.002	-0.09 (-0.23, +0.048) p = 0.19

5.3.5 Does living near opencast coal mining impair health?

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August 1996–February 1999; First Joint Research Programme

Introduction

This study responded to public concern and provided an opportunity to compare exposure to ambient and local PM_{10} sources and health in rural and semi-urban populations and links between them. The aims of the study were:

- to compare the respiratory morbidity of children living close to and some distance from active opencast coal mining sites;
- to compare and characterise particulate levels in these communities using quantitative and qualitative monitoring methods; and
- to investigate the relationship between particulate patterns and respiratory morbidity.

Methods

Five pairs of communities close to (opencast communities, OC) and away from (control communities, CC) active opencast mining sites were compared; pairs were matched for socioeconomic characteristics using Census data and information on urban/rural mix, distance from coast and geography. The study subjects were 4860 children aged 1–11 living within specified geographical areas and registered with a GP.

Exposure was assessed in four stages as follows. (1) Sites and communities were selected using the criteria of community size, distance, intervening dust source, prevailing wind direction and site activity. (2) PM_{10} was monitored and sampled for six weeks in Pairs 1, 3, 4 and 5 and for 24 weeks in Pair 2. In all, 14 609 paired readings of 30 min PM_{10} were recorded (91% data capture). (3) A total of 152 weekly samples from communities and site boundaries were analysed for particle type, particle number, size, shape and the chemical composition of the soluble and insoluble fraction. More than 15 000 (≥100 for each sample) particles representative of total samples were analysed. (4) Indicative opencast enrichment factors, based on sample weight and the proportion of shale particles, were calculated.

Information on previous and current health and lifestyle factors was collected by a postal questionnaire (n = 3216, response rate 68%). For 1 year prior to the study and for the five 6 week study periods, 9980 GP consultations of 2442 children were abstracted from the records of 193 GPs. In total, 59 010 person-days of information on ill-health episodes were collected from 1405 children who completed a daily diary for 6 weeks.

Descriptive and analytical techniques focusing on the comparison between OC and CC were used to analyse the data.

Results

Patterns of PM_{10} levels over time were similar in OC and CC, indicating regional influences, an observation reinforced by comparison with automatic urban network (AUN) data. The geometric mean PM_{10} level was 17.0 µg/m^3 in OC and 14.9 µg/m^3 in CC. Opencast communities were 14% dustier (mean ratio OC/CC 1.14, 95% CI 1.13–1.16). PM_{10} levels followed a rural diurnal pattern. No link between elevated PM_{10} and wind speed, wind direction, or permitted working hours was found. The proportion of shale in boundary and OC samples identified the opencast site as a likely contributor to the additional PM_{10} load in adjacent communities. Flyash, nitrates and sulphates showed mainly regional influences, whereas soot, biological carbon and minerals (including shale) reflected local patterns. Of the weekly samples, 19/35 showed opencast impact based on the weight/shale-proportion enrichment factors. Pairs of communities were similar, overall, in socioeconomic and lifestyle indicators; the sample was representative of the underlying population. Little evidence was found for an increased cumulative or period prevalence of asthma, wheeze, bronchitis or other respiratory illnesses prior to the monitoring period; the asthma prevalence was 21% in OC and CC. Asthmatic children in OC did not have

more, or more severe, asthma attacks. Parent-reported daily respiratory symptoms were similar in OC and CC. The overall consultation rate with GPs was similar in OC and CC, but children in OC were more likely to consult for respiratory, skin and eye conditions (2.1 vs 1.5 consultations per child per year, OR 1.4, 95% CI 1.15–1.70 for pairs 1–4).

Conclusions

Children in OC were exposed to 14% (2 µg/m^3) more PM_{10} than children in CC. Permitted site working hours, wind direction or speed were not linked to this elevation in PM_{10} exposure, but shale, indicative of opencast mining activity, was a measurable contributor. No link was found between living in OC and asthma or other respiratory illnesses, but GP consultations for respiratory, skin and eye conditions were a little higher in OC.

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5.4 Outdoor air pollution mix and health

5.4.1 Effects of air pollution on health in London: Consistency across outcomes

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April 1996–December 1999; First Joint Research Programme

Introduction

The aim of the study was to investigate whether air pollution has short-term effects on daily number of deaths, emergency hospital admissions, visits to A&E departments and GP consultations in Greater London. Parallel time-series analyses of these four health outcomes offer the possibility of examining the consistency of pollutant effects across various outcomes. The same method of analysis was used for each outcome: i.e. Poisson regression to model daily counts of the health outcome in relation to daily measures of air pollution, controlling for meteorological conditions, secular trends, overdispersion and serial correlation. All series were for Greater London for the period January 1992 to December 1994. Pollutants investigated were BS, PM_{10}, NO_2, ozone (O_3), sulphur dioxide (SO_2) and CO.

Associations between air pollution and each health outcome have been reported previously (Atkinson *et al.*, 1997, Bremner *et al.*, 1997, Hajat *et al.*, 1997; Atkinson *et al.,* 1998, 1999; Bremner *et al.,* 1999). This paper focuses on the consistency of results across health outcomes.

Results

Consistent positive associations were found between respiratory events (GP and A&E visits, hospital admissions and mortality) and daily levels of SO_2 and PM_{10} (Table 5.4.1a). These associations were typically a 1–4% increase in the mean daily number of events for a 10th to 90th percentile increase in pollutant. Another consistent finding was the lack of association between respiratory events and O_3. Visits by adults aged between 15 and 64 years to GP and A&E departments and emergency hospital admissions for asthma were all positively associated with NO_2 in addition to SO_2 and PM_{10} (Table 5.4.1b). These associations were of comparable order of magnitude across the health outcomes but did not achieve statistical significance for SO_2. There was also a consistent trend towards stronger associations for asthma in the warmer months (defined as April to September) compared with the cooler months of the year (October to March).

Data for visits to A&E departments for cardiovascular complaints were not available; however, we found that admissions and deaths among those 65 years of age and over were positively associated with all pollutants, significantly so for O_3 (Table 5.4.1c).

Comment

These results suggest that there is some degree of consistency between daily levels of outdoor air pollution and health end-points for both respiratory and cardiovascular disease. This is perhaps unsurprising given the interrelationships between these events but also indicates a degree of coherence in the evidence that strengthens the argument for causality. In further comparisons we shall compare lag structures across the various pollutant/outcome associations.

Table 5.4.1a Associations between respiratory events and air pollution

	NO_2	O_3	SO_2	CO	BS	PM_{10}
GP	1.0 (-0.3, 2.4)	-2.3 (-4.3, -03)	2.2 (0.8, 3.6)	1.4 (0.2, 2.7)	-1 (-2.1, 0.2)	1.5 (0.1, 3.0)
A&E	1.2 (-0.6, 3.0)	1.7 (-0.9, 4.3)	2.8 (0.7, 4.9)	0.8 (-0.8, 2.4)	1.6 (-0.2, 3.5)	3 (0.8, 5.2)
Admissions	1.6 (0.1, 3.2)	1.2 (-1.0, 3.5)	2 (0.3, 3.8)	0.7 (-0.6, 2.1)	1.2 (-0.4, 2.7)	3 (1.1, 4.9)
Mortality	2.3 (-0.4, 5.0)	-3.6 (-7.7, 0.8)	3 (-0.1, 6.2)	2 (-0.3, 4.5)	3 (0.4, 6.0)	4 (0.9, 7.3)

Figures are the percentage change in mean daily number of events associated with a 10th to 90th percentile increase in pollutant (95% confidence limits)

Table 5.4.1b Associations between asthma events in those aged 15–64 years and air pollution

	NO_2	O_3	SO_2	CO	BS	PM_{10}
GP	3.0 (-0.2, 6.3)	4.8 (0.1, 9.7)	3.6 (-0.6, 8.0)	0.9 (-2.1, 4.1)	1.6 (-2.4, 5.8)	5.4 (1.6, 9.2)
A&E	4.4 (0.1, 8.9)	-4.1 (-10.4,2.6)	4.2 (-0.5, 9.1)	4.1 (0.5, 8.5)	5.1 (0.7, 9.8)	7.8 (2.8, 13.1)
Admissions	5.1 (0.8, 9.5)	2.1 (-4.8, 9.6)	4.6 (-0.2, 9.6)	4.0 (-0.2, 7.9)	2.4 (-2.0, 7.0)	5.7 (0.7, 11.0)

Figures are the percentage change in mean daily number of events associated with a 10th to 90th percentile increase in pollutant (95% confidence limits)

Table 5.4.1c Associations between admissions and deaths from cardiovascular disease and air pollution

	NO_2	O_3	SO_2	CO	BS	PM_{10}
Admissions	1.4 (0.2, 2.5)	2.3 (0.2, 4.6)	1.6 (0.2, 2.9)	1.3 (0.2, 2.4)	1.9 (0.6, 3.2)	1.9 (0.6, 3.3)
Mortality	2.3 (0.7, 3.9)	3.5 (0.5, 6.7)	0.8 (-1.0, 2.7)	1.4 (-0.1, 3.0)	1.7 (-0.2, 3.7)	1.9 (0.2, 3.6)

Figures are the percentage change in mean daily number of events associated with a 10th to 90th percentile increase in pollutant (95% confidence limits)

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5.4.2 The short-term health effects of very fine particulate and acid aerosol air pollution upon lung function and respiratory symptoms of asthmatic and non-asthmatic primary-school children: A panel study

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April 1996–March 1998; First Joint Research Programme

Aims

To determine whether:

- daily variations in particulate air pollution are associated with respiratory symptoms and changes in lung function in UK primary-school children;
- children with pre-existing wheezing are more susceptible to any adverse health effects observed; and
- particle mass measures that include a finer size fraction alone ($PM_{2.5}$) provide a better index of public health effect in this age group than those that include more coarse material (PM_{10}) and whether measures of particle acidity improve that index.

Methods and analysis

All children in participating schools aged 9 years by September 1996 were eligible. Previous illness, respiratory symptoms and household factors (smoking, pets, dampness, cooking/heating fuels) were identified by parental questionnaire. Spirometry, exhaled CO, urinary cotinine and skin prick sensitivities to common aeroallergens were measured.

Each child recorded daily respiratory symptoms, medication use and twice daily PEF during a winter and a summer monitoring period (8 weeks from January to March and May to July 1997). Pollutant monitoring within school grounds provided data on NO_2, NO_x, SO_2, CO, O_3, PM_{10}, $PM_{2.5}$, aerosol acid and individual acid and anion species. Weather and pollen data were also collected.

The approach to the analysis followed the two-stage approach of Korn and Whittemore. To identify confounders for the final models, candidate terms for weather effects, pollen, trend, calendar variables (e.g. day of week, schooldays, weekends) and time-series components were included in best-fit linear regression models for each outcome and pollutant on the same day. Final models for each outcome and pollutant (at different lags) could then be applied to each subject and a β coefficient or 'subject-specific effect estimate' generated. These estimates were then pooled (after a weighting related to the inverse of the standard error of that coefficient) to produce overall effect sizes, either for the entire panel or for subgroups with potentially different susceptibilities (e.g. children with pre-existing wheeze, atopic children and passive smoke exposed children).

Results

Panel enrolment and baseline testing

Consent was obtained for 162 children (47% female, 61% of school year). Parents reported wheezing in the previous year in 39 (24%, defined as the symptomatic group), hayfever in 39 (24%), eczema in 47 (29%) and one or more household smokers in 79 (49%) cases. Of the children's homes, 20 (12%) were reported as 'damp', 127 (79%) had central heating, 113 (70%) had gas fires and 121 (75%) used gas for cooking. Only reported hayfever ($p = 0.009$) and damp housing ($p = 0.03$) were significantly more common in the symptomatic group. Spirometry and CO (median 1, range 0–3ppm) were not significantly different between symptomatic and non-symptomatic children. Urinary cotinine was associated with reported household smoking (median 63 ng/mg (range 27–198) vs 31 ng/mg (12–69), $p < 0.0001$ Mann-Whitney U) but not previous symptoms. The number of positive skin tests and a positive test to house dust mite, tree pollen or cat dander were significantly ($p < 0.05$) more common in symptomatic children.

Data collection

An acceptable rate of diary card completion was maintained through the winter (87%) and summer (79%) periods. Four children left owing to school moves during the Easter break and 12 and 11 children, respectively,

were excluded from analysis because of inadequate data collection. Monitored pollutant levels were generally moderate or low, particularly in the summer, owing to unsettled weather (Table 5.4.2a).

Effect estimates from final models

Results for all children show significant adverse relationships between PEF or cough and wintertime particulate and gaseous pollutants as well as nitric acid and sulphate concentrations (Table 5.4.2b). Significant effects were not seen for other symptom outcomes (feeling unwell, short of breath or wheezy) and were reported infrequently and by less than half of all subjects. No significant effects on summer PEF were seen and little pattern was evident for the summer symptom outcomes, which were rarely reported.

Subgroup analysis is under way to determine whether children who are atopic, previously symptomatic or smoke exposed are more susceptible to the effects listed above and to investigate whether such children show other significant adverse effects that are absent when the entire panel is considered.

Table 5.4.2a Median (range) values for pollutant levels averaged across all sites

Pollutant	Winter	Summer	Pollutant	Winter	Summer
NO ppb	12.6 (1–136)	9.8 (1–26)	H^+ ng/m³	8.6 (≤12.7*)	6.3 (≤ 7.6*)
NO_x ppb	31.9 (6–170)	24.8 (5–48)	Cl^- µg/m³	3.0 (0.9–7.3)	0.8 (0.3–5.1)
NO_2 ppb	18.0 (4–35)	13.3 (3–29)	HCl µg/m³	0.3 (0.0–1.7)	0.3 (0.0–1.0)
O_3 ppb	13.0 (2–33)	22 (10–41)	HNO_3 µg/m³	0.5 (0.2–2.2)	1.1 (0.4–3.8)
PM_{10} µg/m³	21.5 (8–46)	18.7 (7–38)	NH_3 µg/m³	5.6 (0.9–23.8)	4.2 (0.6–8.8)
$PM_{2.5}$ µg/m³	12.7 (4–37)	12.3 (5–28)	NH_4^+ µg/m³	2.0 (0.2–15.5)	2.5 (0.5–7.1)
SO_2 ppb	5.4 (2–18)	4.7 (2–10)	NO_3^- µg/m³	3.6 (0.1–29.9)	3.5 (0.7–13.2)
CO ppm	0.5 (0.2–2.0)	0.4 (0.2–0.7)	SO_4^{2-} µg/m³	2.4 (0.8–14.9)	3.8 (1.1–7.8)

* Majority of days (39/56 winter, 47/56 summer) below detectable limit of approximately 5 ng/m³

Table 5.4.2b Significant ($p < 0.01$) results for the complete panel during the winter period

Outcome	Pollutant	Lag	Effect size*	95% CI
Morning PEF	HNO_3	3 day	-0.961	-1.51, -0.42
Afternoon PEF	NO	3 day	-0.310	-0.53, -0.09
	NO_x	3 day	-0.616	-1.03, -0.20
	$PM_{2.5}$	3 day	-0.809	-1.42, -0.20
	SO_2	5 day mean	-1.827	-3.13, -0.52
	SO_4^{2-}	3 day	-0.684	-1.20, -0.17
Cough	O_3	1 day	1.282	1.07, 1.53
	PM_{10}	3 day	1.629	1.16, 2.29

* Either the % change from mean PEF or the factor change in symptom odds per log pollutant rise

5.4.3 Chronic respiratory health effects of cumulative air pollution exposure: A national birth cohort study*

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December 1995–December 1996; First Joint Research Programme

Introduction

This project aimed to investigate the relationship of past and current air pollution exposure to lower respiratory tract illness during childhood, chronic respiratory symptoms in young adults, ventilatory function in young adults and allergic disease in children and young adults. Measures of outdoor air pollution were linked to the areas of residence of members of the British 1958 birth cohort followed by periodic interview from birth to age 33, including a sample examined clinically at age 34–35. The project was funded from December 1995 for 12 months and is now completed.

Methods

Average concentrations of BS and SO_2 monitored in major cities and county boroughs during 1962–1963 were linked to 4502 cohort members; cumulative exposure to BS and SO_2 was estimated for 2352 subjects who had lived in urban areas throughout childhood; and recent annual mean levels of NO_2, SO_2, PM_{10} and O_3 were estimated for 10 km^2 grid squares and linked by postcode to the areas of residence in 1991 of 4971 cohort members followed from birth, of whom 1080 performed spirometry in the home in 1992–93. Outcomes analysed included asthma, wheezy bronchitis and pneumonia in childhood; respiratory symptoms (including cough, phlegm, wheeze and hay fever) at age 33; and spirometric indices (FEV_1, FVC and FEV_1/FVC ratio, before and after salbutamol) at age 34–35.

Multiple logistic regression analysis was used to adjust the effects of pollution for potential confounders.

- For outcomes up to age 16: father's social class, maternal smoking in pregnancy, family size
- For outcomes at age 33: all the above plus own social class, and smoking history by age 33
- For analyses of current pollution: all the above plus area of residence in childhood

Logistic models were used to generate two alternative ways of expressing the pollutant effects.

- Comparing the highest and lowest third of the pollutant distribution
- Fitting a linear trend and expressing the slope as the relative increase in risk (OR) between the 10th and 90th percentiles of the distribution of values for the specific pollutant in question

Results

The results are broadly consistent across all measures of past and current pollutant exposure, and all disease outcomes considered. Despite the large number of potential comparisons, none of the pollutant–disease associations are statistically significant at the 5% level, and in many cases, pollution is associated with a reduced prevalence of symptoms. The effects related to pollution exposure are generally similar before and after adjustment for multiple potential confounding variables, suggesting that it is unlikely that any factors that were not controlled in the analysis would influence the results greatly. The upper 95% confidence limits of the adjusted ORs in analyses of current pollution levels rarely exceed 1.5. The analyses of effects of past pollution exposure are based on smaller numbers (i.e. cohort members brought up in urban areas) but the upper confidence limits are generally less than 2.0. The analyses based on cumulative pollutant exposure are based on the smallest subset, with correspondingly wider confidence intervals, which often include 2.0.

Conclusion

The linkage of past and current outdoor pollution data to the British 1958 cohort has presented a rare opportunity to investigate chronic effects of air pollution exposure. No statistically significant associations were found between any measure of past or current outdoor air pollution exposure and respiratory illnesses in childhood, on symptoms or lung function in adult life.

* Completed by April 1998, abstract presented at second annual review meeting

There was little evidence of confounding of the weak pollutant effects by social and family factors, nor by current (active) or past (parental) smoking. Although some of our analyses had limited statistical power, we conclude that lifetime exposure to outdoor air pollution is unlikely to be an important influence on geographical variations in prevalence of symptomatic lung disease or impaired ventilatory function among young adults in Britain today.

The results must be interpreted in a historical context and generalised with caution to earlier and later cohorts. This generation was exposed as young children to high levels of urban smoke and SO_2 pollution, mainly due to domestic coal smoke. Concentrations of these pollutants decreased as the cohort moved through adolescence and adult life. Current pollution exposure, although at lower levels, is of a different nature, with a sizeable component attributable to primary and secondary pollutants related to motor traffic. Our results relating to childhood illnesses may not be relevant to children growing up today.

5.5 Traffic and health

5.5.1 The effect of exposure to motor vehicle traffic at home on the prevalence of wheeze in Nottingham school children: A spatial analysis using a geographical information system

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September 1997–September 1998; First Joint Research Programme

Introduction

Vehicle exhaust emissions have steadily increased over recent years in the UK (DoE 1992), but their role in causing or exacerbating asthma remains unclear. We have previously carried out studies of wheeze in Nottingham school children and found no evidence of an association with level of vehicle traffic activity near the child's school at an ecological level, and a relatively weak association at an individual level for primary-school children only. The study has now been extended to investigate the contribution of vehicle exhaust exposure at home on the occurrence of wheeze, using two indicators of exposure, distance of home from a main road and traffic activity near the home. The primary aim was to determine whether there is an association between proximity of the child's home to a main road and wheeze in the past year, but in particular to look at this relation among those living fairly close to a main road. This is because evidence suggests concentrations of oxides of nitrogen (NO_x), CO, particulates and non-methane hydrocarbons are highest at the kerbside and decrease with distance from the road, with little decline beyond about 60 m (DoT 1993). A further aim was to establish the independent effect of traffic activity in the vicinity of the home on the occurrence of wheeze using an existing Traffic Activity Index (TAI) map of school exposure (generated in previous collaboration with Margaret Bell).

Methods

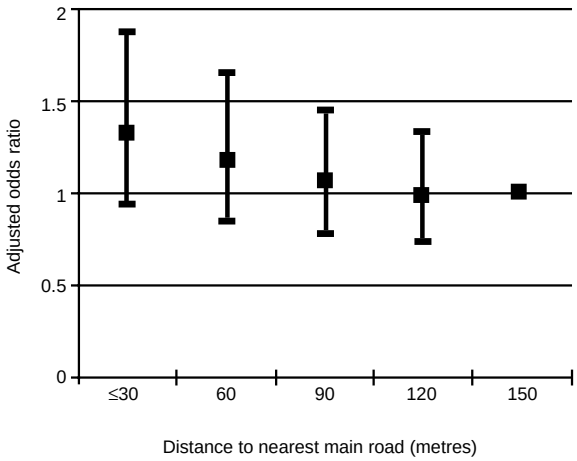
A case–control set of 6576 primary-school children and a random sample of 3894 secondary-school children, for whom extensive questionnaire information is already available on age, sex, early life factors (birth weight, maternal age, number of siblings, prematurity, breastfeeding and parental smoking), eczema, hayfever, and home postcode, were used for this investigation. The outcome variable was parent-reported wheeze in the past year.

To estimate home exposure to road vehicle traffic, the child's residential postcode was converted into a six figure grid reference (1 m resolution) using Ordnance Survey Data-point. A Geographical Information System (GIS) (Arc/Info PC) was used to plot the postcode locations, link these data with a digitised map of all A and B roads and motorways in the Nottingham area (40 km by 40 km tile), and hence compute the shortest distance to a main road for each point. This new variable was then linked to the existing datasets in SPSS. Analyses were carried out on primary- and secondary-school children separately. The distance variable was initially analysed in quartiles to explore the relationship between distance and current wheeze, and the statistical significance of the association was assessed using the chi-squared test. The relationship was then explored among those living relatively close to main roads since this is where most of the effect is expected to be. All those living within 150 m of a main road were selected, and the distance variable was analysed in 30 m bands, again using chi-squared to test for an association. It was not appropriate to analyse in smaller bands since very few children were assigned a value of less than 20 m for the distance variable. Traffic flows had been measured previously on roads in and around Nottingham and a TAI had been computed for each 1 km^2 gridsquare of a map of the area containing a school. To estimate the level of traffic flow near the home, each child's home grid reference was linked to this map, and assigned the value of TAI for that gridsquare where possible. This variable was then analysed in quartiles. To adjust all analyses for potential confounders such as social deprivation, maternal smoking and early life factors, multiple logistic regression techniques were used in SPSS.

Results

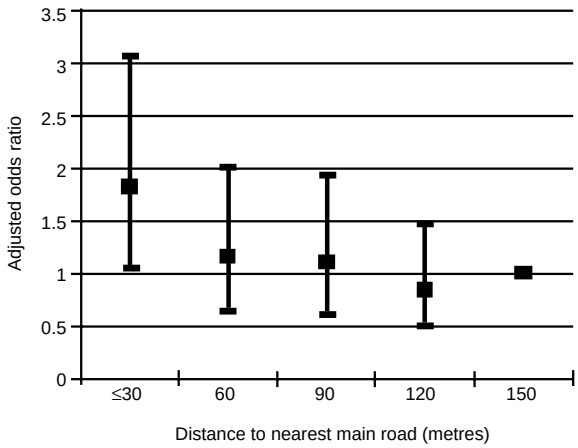
Home postcodes were successfully mapped for 93% (6147) of the primary case–control dataset, of which 2483 were cases, and for 96% (3722) of the secondary-school sample. For the primary-school analysis, the median distance from the home to a main road was 321 m, with an interquartile range of 149–700 m. When analysed as quartiles, there was no difference in wheeze prevalence across the quartiles (p_{trend} = 0.97). However, when the association for distances less than 150 m was explored in more detail (n = 1541), there was a clear decline in risk with increasing distance from a road, which remained after adjustment for age, sex, social deprivation (Carstairs score) and maternal smoking (Figure 5.5.1a). This association was of borderline significance when fitted as a linear trend through the categories (p = 0.06), although the decline appeared to flatten off beyond about 100 m. The adjusted OR for living within 30 m of a main road relative to 150 m was 1.29 (95% CI 0.98–1.70).

Figure 5.5.1a Adjusted odds ratios and 95% confidence intervals for wheeze in the past year in relation to distance of home from main road in primary-school children



The results for secondary-school children showed the median distance was 372 m (interquartile range 163–742 m), and again no trend was seen across the quartiles (p_{trend} = 0.64). For those living within 150 m of a main road (n = 849), however, there was a clear decline in wheeze risk with increasing distance from a road, which was significant when fitted as a trend and remained after adjustment (p = 0.04; Figure 5.5.1b). For secondary-school pupils, the adjusted OR for living within 30 m of a main road relative to 150 m was 1.74 (95% CI 1.15–2.63). Further adjustment for breastfeeding, maternal age and premature birth had little influence on the relationships.

Figure 5.5.1b Adjusted odds ratios and 95% confidence intervals for wheeze in the past year in relation to distance of home from main road in secondary-school children



A home TAI was assigned to 5741 primary school children and 3355 secondary-school children, and was not seen to be associated with wheeze in the past year (p = 0.84 and 0.77, respectively, for trend across the quartiles).

Conclusion and discussion

These findings suggest there is an inverse relationship between distance from home to nearest main road and the risk of wheeze, but only up to distances of about 100 m. The nature of the relationship, especially for secondary-school children, resembles that seen when concentrations of pollutants are plotted as a function of distance from a road (DoT, 1993). Our findings are unlikely to be due to confounding since we have allowed for the main potential confounders. Since we only had postcodes and not full addresses of our subjects, there may be some appreciable degree of error in estimating distance from roads, but this is unlikely to be systematic and will have tended if anything to result in a weakening

of the observed relationship. Traffic activity in the proximity of the child's home was not seen to be a risk factor for wheeze, possibly because the TAI variable used is subject to large random error and also is a measure of exposure for a 1 km^2 area, which may not be sufficiently localised. In conclusion, school children living on a main road are at an increased risk of wheeze, and this risk declines with increasing distance from a main road up to about 100 m.

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5.5.2 Relationship of asthma and allergic rhinitis to local traffic density and ambient pollution modelled at a small area level*

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April 1997–March 1998; First Joint Research Programme

Introduction

This project aimed to investigate small area variations in the prevalence and severity of wheezing illness and other allergic disorders among children in Sheffield, and their relationship to estimates of exposure to traffic-related air pollution.

Methods

In 1991, all children aged 11–16 attending secondary schools in Sheffield were included in a questionnaire survey of symptoms related to asthma, allergic rhinitis and eczema. Questionnaires were sent to the parents of 23 054 children; 18 203 questionnaires were returned (response rate 79%). Postcodes of residence and schooling were derived for 17 998 (99%) respondents. This study was restricted to 14 823 children born 1975–1980 living within Sheffield City boundaries. Postcodes were used to derive grid references for exposure estimation, and to assign a deprivation score (Carstairs index) to each child based on their area of residence. More detailed information on the home environment was available for 1427 children, who were recontacted in 1993 as part of a case–control study of more severe asthma.

Exposure to traffic pollution was estimated by three methods using Geographical Information Systems techniques:

- proximity of home and school to the nearest main road;
- traffic flows along major roads within a 150 m radius of home and school, including an index of heavy goods vehicle (HGV) traffic; and
- estimates of pollutant concentrations at place of residence and schooling based on pollution modelling.

The pollution estimates were validated against ambient NO_2 levels measured at 2 monthly intervals by passive diffusion samplers at selected sites close to and distant from roads in the Sheffield area during 1997–1998.

Small area variations in symptom prevalences were assessed by map smoothing techniques. Relationships of respiratory symptoms to estimated pollution exposure were analysed at the individual level by logistic regression, adjusting for age, sex and deprivation score.

Results

There were 2314 children with a history of wheeze in the last year, of whom 659 had severe symptoms (speech-limiting and/or frequent attacks). A total of 2257 children had symptoms of allergic rhinitis. Over half (1325/2314) of the wheezers who also had rhinitis or eczema ('atopics') were analysed separately from those who did not. No substantial or significant associations were found between any measure of local traffic density or vehicle-related pollution exposure and any symptom outcome or subcategory. Illustrative OR estimates and 95% CIs, adjusted for age, sex and deprivation score are shown in Table 5.5.2a.

Discussion

This large study, in a large urban area where the local topography tends to enhance variations in traffic-related pollution, offers no support for the hypothesis that the prevalence of childhood asthma or allergic rhinitis is substantially increased by living close to busy roads. More specifically, we found no evidence of an adverse effect of HGV traffic, nor of estimated outdoor exposure to vehicle-related pollutants. The reason why studies in other countries have found such relationships remains unclear, but our results argue against a causal relationship between local traffic exposure and the development of childhood asthma. This study within a single urban area does not, however, exclude a possible risk from regionally distributed secondary pollutants such as O_3.

* Completed by April 1998, abstract presented at second annual review meeting

Table 5.5.2a Odds ratio estimates for measures of local traffic density and vehicle pollution exposure and asthma and allergic rhinitis (95% CI)

Measure	Recent wheeze	Severe wheeze	Atopic wheeze	Allergic rhinitis
Nearest main road to home (<50 m vs >150 m)	1.08 (0.97–1.22)	1.08 (0.85–1.38)	0.99 (0.80–1.22)	0.89 (0.79–0.99)
Per 100 m of main road within 150 m of home	1.00 (0.97–1.04)	1.01 (0.96–1.08)	1.00 (0.96–1.06)	0.94 (0.91–0.98)
Per 1000 km non-HGV traffic flow within 150 m	1.00 (0.98–1.02)	1.02 (0.98–1.06)	1.01 (0.97–1.04)	0.96 (0.94–0.98)
Per 1000 km HGV traffic flow* within 150 m	0.64 (0.31–1.31)	0.46 (0.13–1.61)	1.27 (0.44–3.70)	1.11 (0.51–2.38)

* Adjusted for total traffic flow

5.5.3 The effect of relieving traffic congestion on pollutant exposure and respiratory morbidity: The Bypass Study

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February 1996–December 1999; First Joint Research Programme

Introduction

The Deeside Road Link (A548), which was opened in March 1998, bypasses Shotton and Connah's Quay in Clwyd, North Wales. The aim of our project is to investigate the effect of relieving traffic congestion on pollutant exposure and respiratory morbidity, and to investigate whether respiratory health and air pollution improve when people cease to be exposed to high levels of exhaust fumes.

Methods

Congested and uncongested streets in Shotton and Connah's Quay in Clwyd were identified and subjects recruited from these areas. A respiratory survey and pollution measurements were conducted in both areas before and after the opening of the bypass.

A questionnaire was sent to all subjects. Pulmonary function tests and skin prick tests were conducted on subjects over 7 years old, while serum IgE measurements were made on subjects over 18 years old. The aerosol concentrations in two size ranges, fine and coarse modes, were determined using a dichotomous sampler. Personal exposure to NO_2 and CO was monitored using diffusion badges.

Results

The congested streets have on average 30% more particulate pollution than uncongested streets. The largest differences between the two sites occurred during days of high traffic flow and low wind speeds. The fine mode is dominated by sulphate and ammonium, while sodium, magnesium, potassium and calcium constitute on average 5% of fine mode mass. An association between increases in pollutant levels and traffic counts has been observed. Significant differences in pollution levels in the congested and uncongested streets, before and after the opening of the bypass, have been observed.

6 Public health impact

6.1 Perception and communication

6.1.1 Public perceptions of risks to health from air pollution and views on air quality information

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October 1996–March 1999; First Joint Research Programme

Introduction

This study explored public views about the risks to health from air pollution, and public access to, and views on, air quality information.

The study took place in Teesside and Sunderland. Teesside is characterised by Britain's largest steel and petrochemical complex with approximately 12 000 people living within 1 km of industry. By contrast, most heavy industry in Sunderland (mainly shipbuilding and coal-mining) has now ceased. Local Authority policy on air quality bulletins differs in the two areas. Teesside produces regular, local air quality bulletins for the public, which are disseminated through different media, whereas Sunderland does not.

Methods

The study involved five communities varying in their geographical proximity to industry and on the basis of socioeconomic indicators. A postal questionnaire survey was made of 5000 adults (response rate 62%) followed by in-depth interviews with 40 respondents (with and without illnesses affected by air pollution).

Results

Perceptions about air pollution and health issues did not differ by sex. Proximity to industry, residence in Teesside, socioeconomic position and presence of a chronic illness were significantly associated with regarding local air pollution as a serious problem; none of these factors were associated with viewing traffic pollution as a serious problem. Views about whether air pollution affected a range of illnesses (including asthma, bronchitis and lung cancer) did not differ by proximity to industry, age, sex, chronic illness and socioeconomic status. However, respondents who suffered from respiratory illnesses were significantly more likely to feel that air pollution affected their illness (e.g. asthma affected by air pollution, OR 5.57, 99% CI 2.27–13.68). They were also more likely to feel that smoking affected their illness, but to a lesser degree (e.g. asthma affected by smoking, OR 2.30, 99% CI 1.37–3.87). Although respondents living closest to heavy industry were not more likely to believe that they personally suffered from an illness affected by air pollution, they were significantly more likely to believe that other members of their community suffered from illnesses affected by air pollution (OR 0.27, 99% CI 0.17–0.43). Rating local air quality as 'poor' or 'very poor' was associated with proximity to industry, residence in Teesside, chronic illness and those aged under 65. No differences were found for questions about city centre air quality in either Teesside or Sunderland, suggesting that although local experiences of air quality differed, views about air quality elsewhere were similar.

Over 90% of survey respondents felt that making air quality information available to the public was 'important' or 'very important', although only 12–21% (Sunderland) and 40–51% (Teesside) had actually seen such information. Socioeconomic status, residence in Teesside and being aged over 65 were significantly associated with having seen this information. The proportion of respondents who stated they had come across air quality information ranged from 28–19% (Teesside) and 21–10% (Sunderland).

The in-depth interviews added a further dimension to this study by providing detailed insights into views on, and experiences of, air quality. In particular, they highlighted differences between neighbourhoods and how the impact of air pollution is differentially distributed and experienced. Different ideas about ill-health causation emerged. For example, concern with an increase in childhood asthma was expressed in all communities. However, Sunderland interviewees and those in the communities distant from industry in Teesside did not associate their own asthma or the increase in childhood asthma in their particular community with air pollution. Other factors such as stress, genetics and pollen were viewed as more important. The interviewees from these communities tended to adopt strategies to distance themselves and their community — both geographically and socially — from the risks posed by industrial air pollution. In direct contrast, those interviewees living close to industry felt that the increase in childhood asthma in their community was due to industrial air pollution.

Of those interviewees who had come across air quality information, most had not found it to be helpful or useful, either because it was not local enough or because they felt it was only for those who suffered from an illness affected by air pollution or those who lived in areas of particularly poor air quality. In Teesside there was also a sense of mistrust surrounding air quality information.

Conclusions

These findings indicate that perceptions about the quality of the local environment are strongly influenced by personal experience of air pollution. Proximity to heavy industry and chronic illness are most strongly associated with views about air quality. Making air quality information available to the public is regarded as important, but current local and central government practices do not appear to be providing information that is useful. Further qualitative research (possibly involving focus groups) would be a useful first step in identifying more useful air quality information strategies.

During April 1999–October 1999 the findings from this research will be disseminated to a wide range of audiences including national government, local authorities, policy-makers, those studying issues relating to perceptions of air quality and those involved with the provision and presentation of air quality information.

6.1.2 Asthma and air quality: A survey of schools and young people[a]

L Kendall

National Foundation for Educational Research, Slough, Berkshire

April 1996–July 1997; First Joint Research Programme[b]

Aims

This study concerned young people with asthma aged from 11 to 16, and had three main aims:

- to investigate how these young people obtain information on air quality and how they respond to such information;
- to assess the role of parents and schools in providing this information, supporting pupils with asthma and helping them to make more informed choices; and
- to consider how information could be better targeted for this group of young people.

Methods

Questionnaires were completed by over 1800 pupils with asthma, over 80 of whom also took part in group discussions. The representatives of 157 secondary schools and 55 special schools also completed questionnaires.

Pupils were asked about factors that made their asthma worse, and about the sources of information about asthma and air quality that they found most useful.

Results

Both indoor and outdoor air quality affected the majority of the pupils, but their level of knowledge about sources of information about air quality, and their confidence in interpreting it, seemed low.

From the questionnaires completed by school representatives, it was clear that schools varied widely in the support and information they provided to young people with asthma, and that in the majority of schools the formal curriculum did not address links between asthma and air quality.

Four key points emerged.

- Information must be carefully targeted, both in content and format, taking into consideration pupils' ages and interests. The ways in which information is provided must also acknowledge that, while asthma is a common condition, young people do not want it to limit their activities or make them appear to be different from their peers.
- Pupils can make informed judgements about how to respond to variations in air quality only if they have a sufficient understanding of the links between asthma and air quality. Schools could play an important part here, in making these links more explicitly in the curriculum. Some schools may feel that this is not part of their role, or that they do not have the required expertise. Coordination between health professionals, health educators and schools could ensure that young people are helped to understand these links.
- Many pupils find that doctors and asthma clinics are important and helpful sources of information about asthma and air quality. How can health professionals improve the ways in which they communicate with those young people who do not respond so positively? How can they help pupils to integrate factual information and personal experience, so that individual pupils can learn how to respond appropriately to variations in air quality?
- Pupils need to be aware of the wide variety of sources of information that already exist, and ways of increasing levels of awareness of these sources should be investigated.

[a] Completed by April 1998, abstract presented at second annual review meeting
[b] Principal Investigator, Dr W Keys

6.2 Predicting health impacts

6.2.1 Towards assessing and costing the health impacts of ambient particulate air pollution in the UK*

JF Hurley[1], MR Holland[2], A Markandya[3], BG Miller[1], HR Anderson[4], JG Ayres[5], PT Donnan[1,6], RM Harrison[7], K King[2], JR Stedman[2] and KJ Stevenson[2]

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August 1996–October 1998, First Joint Research Programme

Aims

The aims of the study were:

- to assess the feasibility of quantifying and costing the adverse health effects of particulate air pollution in the UK;
- to identify the main determinants of the results; and
- to assess the reliability of the answers.

The focus was methodological, our aim being to quantify as much as practicable, even where information was questionable, and to describe the reliability of the results.

Methods

The study was based on existing data, including:

- measurements and maps of ambient particles in the UK;
- epidemiology, leading to exposure–response (E-R) functions linking particles and health;
- demographic data, including mortality and morbidity rates; and
- economic valuation studies of health effects.

Data were assessed and linked into an integrated assessment scheme, using a Geographical Information System (GIS; ArcInfo). Much of the study was in parallel with, and complementary to, the work of two Department of Health (DH) expert groups, on quantification (QUARG) and on economic assessment (EAHEAP), both of which involved several members of the project team.

We focused principally on quantifying the effects of particles expressed as particulate matter of aerodynamic diameter <10 µm (PM_{10}), assuming no threshold. The most reliable E-R functions (acute mortality, hospital admissions) are as in QUARG. Other effects quantified included: several acute morbidity end-points; development of chronic bronchitis; and 'chronic' mortality, assessed via cohort studies and quantified using life-table methods (see Miller & Hurley, Section 6.2.2). Selected sensitivity analyses included:

- different ways of estimating background morbidity rates;
- alternative E-R functions;
- alternative valuations of a life-year lost (VLYL) — £100 000 (Willingness-to-Pay) and £10 000 (Revealed Preference), with lower values at older ages;
- effects of particles represented as $PM_{2.5}$ or as sulphates;
- the impact of thresholds on PM_{10} damage estimates;
- different discount rates (11% and 3%).

Results

As expected, mortality as estimated from cohort studies was the dominant single effect. The benefits of a sustained PM_{10} reduction were estimated at about two weeks' gain in life expectancy, on average, per µg/m^3 PM_{10}, for those who experience the full benefits. The overall benefits to the current population and to new cohorts in England and Wales were estimated at about £3bn per µg/m^3 sustained reduction in PM_{10} (£60bn for overall PM pollution). There are considerable

* Modified from presentation made at the second annual review meeting of the Joint Research Programmes on Outdoor and Indoor Air Pollution (1998)

uncertainties about this number, which includes both acute and chronic mortality effects.

Acute mortality effects were estimated separately at about 8800 deaths brought forward annually in the UK. The amount of life-shortening is unknown but believed to be small, on average. Associated long-term benefits of a sustained reduction were estimated at between £1.5bn and £4.5bn, i.e. much less than those estimated from cohort studies.

By comparison, hospital admission effects (respiratory, cardiovascular) were small.

Other effects were estimated less reliably. However, results suggest possibly substantial effects of PM on development of chronic bronchitis, days of restricted activity (RADs), various acute effects in asthmatics, and days of symptoms in people generally. Several of these estimates were sensitive to how background rates were estimated. These results are indicative only, given the associated uncertainties.

Results, limited by lack of suitable E–R functions for $PM_{2.5}$ and sulphates, appear not to be sensitive to index of particles used. However, a threshold at 15 $\mu g/m^3$ *daily* PM_{10} would reduce damages to about 30% of their no-threshold value; at 25 $\mu g/m^3$, values would be one-half as low again.

Conclusion

Even when there are major underlying uncertainties, substantial progress can be made towards quantifying and costing the health effects of ambient PM_{10}. In particular, it has been helpful to include a wide range of health end-points, including mortality assessed from cohort studies. Major assumptions were sometimes necessary, based on limited evidence. Sensitivity analyses have helped in understanding their importance. Quantification and costing would be improved by better estimation of background morbidity rates; fundamental valuation studies; better epidemiology of chronic morbidity and milder acute effects (RADs, symptoms); and better knowledge of a threshold, or not. These are suggested priorities for further research. In particular, we think that better investigation and representation of uncertainty would help in practical application of the methods and results to quantified analysis of public policy issues, although the work to date can already be helpful.

6.2.2 Quantifying chronic mortality impacts of pollution changes*

BG Miller and JF Hurley

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August 1996–October 1998; First Joint Research Programme

Introduction

Cohort studies have compared mortality in cities with different levels of airborne particulate pollution (Dockery *et al.,* 1993; Abbey *et al.,* 1995; Pope *et al.,* 1995). They provide strong evidence for a relationship between pollution and mortality rates, and supply exposure–response coefficients.

Predicting impact on chronic mortality

Mortality hazard rates (age-specific death rates) vary with age and may also vary with time. It is useful to arrange a set of hazards as in Table 6.2.2a. Then standard life-table calculations down the diagonals (shaded) convert these to survival probabilities and, hence, for a given population with age distribution e_i, to numbers of deaths by age and year. A number of assumptions are required, about how death rates will change (or not) in the future, and about birth rates b_j.

Summarising mortality experience

Thus predicted mortality can be calculated for any set of hazards, and scenarios with an assumed reduction in pollution, and associated drop in hazard rates, can be compared with a no-change scenario. Of the many summary measures of impact possible from the life-table calculations, some of the most useful are:

- change in expected years of life (from birth or from age when change is introduced);
- change in numbers of deaths occurring in a period;
- change in years (or 'person-years') of life lived: YOLL.

These can be summarised over any subpopulation or any time period.

Valuation of mortality impact

Deaths or years lived may be assigned economic values, which may also differ by age and/or by time (e.g. by discounting). Thus predicted impacts can be converted to economic valuations.

Assumptions made

Many simplifying assumptions have to be made in creating the baseline and impacted hazard tables. For example:

- future hazards will be the same as current;

Table 6.2.2a Schematic arrangement of hazard rates for life-table calculations

Age	**Entry**	**Year**														
		1995	**1996**	**- - - -**	**1999**	**2000**	**2001**	**2002**	**- - - -**	**j**	**- - - -**	**2103**	**2104**	**2105**	**- - - -**	**2135**
		Births	**b_1**	**- - - -**	**b_5**	**b_6**	**b_7**	**b_8**	**- - - -**	**b_j**	**- - - -**	**b_{108}**	**b_{109}**	**b_{110}**	**- - - -**	**b_{140}**
0	e_0	h_0	h_0		h_0	h_0	h_0	h_0		h_0		h_0	h_0	h_0	h_0	h_0
1	e_1	h_1	h_1		h_1	h_1	h_1	h_1		h_1		h_1	h_1	h_1	h_1	h_1
2	e_2	h_2	h_2		h_2	h_2	h_2	h_2		h_2		h_2	h_2	h_2	h_2	h_2
ı																
i	e_i	h_i	h_i		h_i	h_i	h_i	h_i		h_i		h_i	h_i	h_i	h_i	h_i
ı																
103	e_{103}	h_{103}	h_{103}		h_{103}	h_{103}	h_{103}	h_{103}		h_{103}		h_{103}	h_{103}	h_{103}	h_{103}	h_{103}
104	e_{104}	h_{104}	h_{104}		h_{104}	h_{104}	h_{104}	h_{104}		h_{104}		h_{104}	h_{104}	h_{104}	h_{104}	h_{104}
105	e_{105}	h_{105}	h_{105}		h_{105}	h_{105}	h_{105}	h_{105}	h_{105}	h_{105}		h_{105}	h_{105}	h_{105}	h_{105}	h_{105}

* This work was supported by DH. The methods have been used in work commissioned by National Power and in the European ExternE project.

- future birth rates will be the same as current;
- net migration has a null effect.

These assumptions could be relaxed on the basis of expert prediction. In addition, the construction of the hazard table in two dimensions allows great flexibility in accommodating the impact. Possibilities include:

- multiple or phased pollution changes;
- full impact of pollution phases in gradually;
- impact differs by age.

Different set of assumptions and conditions can be postulated. The simulations can be run for various combinations, by which it is possible to quantify the sensitivity of the results to changes in the assumptions.

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7 Discussion

7.1 Review of the Joint Research Programmes

The Joint Research Programmes have been designed to add to the current state of knowledge on the links between air pollution and ill-health and thereby to facilitate development of UK Government policy.

The third annual review meeting of the Joint Research Programmes on outdoor and indoor air pollution provided an opportunity to review the progress of studies undertaken in the two programmes and, in so doing, to assess the contribution the programmes have made to increased understanding about air pollution and health. The meeting also provided an opportunity to discuss developing research areas in the UK and elsewhere in Europe and, in particular, to propose new research to help advance the development of UK Government policies on air pollution.

This section reflects the various discussions held over the three days of the meeting, for each of the major topic areas — exposure and modelling, mechanisms, volunteer studies, epidemiology and public health — and concludes with a discussion of the policy implications of the research and some recommendations for future work.

7.2 Exposure and modelling

7.2.1 The contribution of the Joint Research Programmes to research on exposure monitoring and modelling in the UK and internationally

Studies on exposure measurement in the Joint Research Programmes contribute to several areas of developing UK and international research on air pollution; some key areas are described below.

Environment and health research for Europe

The European Science Foundation (ESF), European Community (EC) and the World Health Organization (WHO) have jointly established research priorities for Europe (presented to the Third Ministerial Conference on Environment and Health, London, June 1999, http://www.who.dk/london99/). Recommendations relevant to the Joint Research Programmes on outdoor and indoor air pollution include the following: research into mechanisms pertinent to the effects of short-term and long-term exposure to particles and to the air pollution mix, with particular emphasis on physical and chemical characterisation; research to improve the understanding of the effects of long-term exposure to particles; and research into the role of biological contaminants, especially in the exacerbation of allergic effects and asthma. In the latter case in particular, there is an important role for exposure assessment.

Exposure determinants

Understanding the determinants of exposure to air pollutants is important for making policy on their control. For example: how much can indoor exposure in the home be attributed to specific activities such as cooking or vacuuming; how do different types of traffic contribute to different types of air pollution; how is transboundary air pollution affected by different contributions from waste management, landfills, incineration, sewage treatment? Assessment of each of the factors that determine exposure levels in a particular scenario has important implications for policy-making and the introduction of appropriate control measures.

However, there are limitations in the data currently available to assess exposure determinants: for example, although there are, in the Joint Research Programmes, some studies on household characteristics and how these impact on indoor air pollution, this is an area that warrants further investigation; there is currently too great a discrepancy between models for long-range transboundary air pollution and local pollution measurements; and exposure assessment around landfills is also very limited.

Particles

The contribution of the 'personal cloud' to total personal exposure to particles has been a topic of developing research interest. The personal cloud is mostly due to resuspended dust and is therefore of most relevance to indoor exposures to particles. Modelling can be undertaken to take account of the personal generation of dust. It is not clear whether the coarse particulates that largely make up the personal cloud have any significant health impact. When exposure is related to a biological end-point, personal exposure estimates correlate better than local ambient air measurements, which suggests that the possible impacts of the personal cloud cannot be dismissed. However, even if particles related to the personal cloud were ever shown to have an impact on health, there would seem to be little that could be done in terms of control.

The relationship between particles and humidity is not well understood. Particles that are hygroscopic grow in humid conditions, leading to a higher mass concentration. The degree of charge on particles could also be affected by humidity. Although patient complaints appear to be higher on high humidity days, epidemiological studies that include humidity data generally show no correlation between humidity and health end-points.

7.2.2 Future directions for research on exposure to air pollutants

Exposure measurement

Information about exposure to air pollutants will facilitate more effective intervention. It is known that intervention strategies in one location can impact on another; traffic control measures, for example, may improve air pollution in one location to the detriment of that in another location. An understanding of the contributions to total exposure made by different sources will also facilitate the appropriate targeting of control interventions.

Better exposure measures will improve the usefulness of data from epidemiological studies. In order to make the best use of resources, methods for exposure measurement used in exposure studies and those used in epidemiological studies should be coordinated.

Exposure modelling

Exposure modelling may facilitate quantitative interpretation of exposure data, notably for policy development. For example, although with the current time-series studies it is now possible to link pollutant levels directly with health impacts, it is not yet known how much indoor sources may contribute to exposure to total PM_{10} (particulate matter of <10 µm aerodynamic diameter). More quantitative studies may give some clues as to how to control particle levels in indoor air.

Exposure modelling can also be used to assess the impact of measures to modify exposure; for policy considerations, this can be crucial. For example, if controls on vehicles are to be put in place, modelling can be used to predict what the impact on outdoor exposure to air pollutants will be.

Models can be used to determine frequency distributions of exposure. Such information may also be valuable for policy decisions; different control strategies may be appropriate for populations with different exposures. If distributions of exposure could be combined with distributions of sensitivity to a response, in Monte Carlo type analyses, it should be possible to predict effects in a population. Time-series studies can already do this, as by definition they cover a distribution of exposures and a distribution of sensitivities. What is not necessarily clear is what the causal association between exposure and response may be.

Modelling of exposure could enhance information available from epidemiological studies by, for example, reconstructing the exposure of those who become or became ill. The development of models for exposure could also help in retrospective constructions of the UK air pollution profile (particularly in terms of the pollution mix). The modelling of air pollutant exposure for individuals is still in its infancy, but could provide a very helpful way of investigating health effects.

More collaboration between those developing exposure models and those conducting monitoring studies would facilitate the validation of the models.

Personal exposure

Much progress is now being made in developing techniques for personal exposure measurements, and it should be possible to develop these further for use in epidemiological studies. However, personal sampling is not always practical in an epidemiological study; by increasing costs and limiting participation such an approach may limit study power. It is more realistic to use personal exposure measurements to validate exposure modelling. It will be important to link personal estimated exposure levels with external determinants of pollution sources, as it is necessary to know not only how much pollution there is, but also where it comes from.

Particular policy questions will be better addressed by the use of personal exposure monitoring. For example, such techniques would be appropriate in studies to identify where air quality standards should apply, such as a study on the benefits to health of controlling roadside pollution levels. Personal exposure measurements would also be informative in any ‘natural experiment’ or intervention studies.

Concentrating on the development of personal exposure monitoring might seem to imply a continued focus on short-term, rather than long-term exposure. However, there are well-established models for estimating cumulative exposure, which could be used in studies on environmental exposure.

For longer-term studies it is especially important to identify situations where there are contrasts in exposure scenarios; however, this will become progressively harder to achieve as ambient pollution levels become lower. Where there are large differences in outdoor exposure levels for the populations being studied, there is less need for personal exposure monitoring.

Indoor versus outdoor sources

In general, people spend 95% of their time indoors, a high proportion of which is spent in the bedroom. Even this is a simplification; whereas some individuals may only be at home for 60–70% of their time, others may spend 100% of their time at home. Indoor exposure to pollutants has by far the greatest influence on personal exposure overall for most pollutants, including PM_{10}, volatile organic compounds (VOCs) and nitrogen dioxide (NO_2) (but notably not sulphur dioxide (SO_2) or ozone (O_3)). Little is known about indoor exposures in schools and offices, and, within the UK, even data on exposure in the home comes largely from England; much less is known about the situation in Scotland, Wales and Northern Ireland.

Although outdoor air pollution often drives indoor air pollution, there are notable exceptions and, for example, the data available are not yet sufficient to allow determination of whether it is the indoor or the outdoor environment that dominates PM_{10} exposure. Certainly, in the case of PM_{10}, but not nitrogen dioxide or volatile organic compounds, outdoor sources may have a major effect in influencing indoor levels of these pollutants. Characterisation of particles will help to identify whether the important determinants of PM_{10} exposure in indoor air are from indoor or outdoor sources. Certainly there

are sources of particles in the home, and the impact these sources may have on health cannot be measured in time-series studies, which reflect only day to day changes in outdoor concentrations.

Modelling of exposure determinants, studies on the influence of outdoor peaks on indoor levels, and characterisation of particles may all help to improve understanding of the role of particles in the indoor environment.

7.3 Mechanisms

7.3.1 The contribution of the Joint Research Programmes and directions for future research into mechanisms

Little work on the mechanisms of action of particles has been undertaken under the existing Joint Research Programmes. More research is needed in several areas: on the characterisation of particles, both composition (e.g. the importance of transition metals, endotoxins, polycyclic aromatic hydrocarbons (PAHs), primary and secondary particles, acidity) and size (e.g. the relative importance of PM_{10}, $PM_{2.5}$, ultrafine); on the origin of particles (investigations of days when primary particles predominate over secondary; trajectory studies); on the role of co-exposures (which has scarcely been looked at for particles); and to investigate pathways of toxicity, including the possibility of common pathways (e.g. transcriptional activation; oxidative stress) for different end-points.

Characterisation

Work on the characterisation of particles is still in the early stages. The qualitative observations made so far (e.g. BéruBé & Richards, Section 2.5.1; Murphy *et al.,* Section 3.1.1) are certainly of interest but still need to be followed up by quantitative studies. Of particular interest will be studies on the ultrafine fraction. It is still not well understood what the characteristics of particles are that drive their effects; for example, how important is the particle surface? Adsorption at particle surfaces alters their physicochemical and toxicological characteristics. Reduced antioxidant from lung lining fluid could, for example, adsorb onto particles and detoxify them.

More work is needed to define exposures to particles better. In particular, the types of particles used in experimental studies should be defined more precisely, and similar types of particle should be used in different studies, to aid comparison of results. Currently, different studies use different surrogate particles to address different hypotheses, leading to limitations in the

applicability of the studies in general terms. Some surrogates are only components of the total particulate mix, others may be particularly high in certain constituents (e.g. residual oil fly ash (ROFA) and sieved suspended particles, which are high in transition metals). More research is needed to decide the most appropriate methods to generate particles for experimental studies (e.g. the Harvard particle concentration system; the UK Harwell system for generating ultrafine particles of known characteristics).

Pathways of toxicity

Several areas of research into mechanisms of toxicity, particularly of O_3 and particles and the possibility that they might act through common pathways, merit further consideration.

For example, what is the role of the lung lining fluid in the defence against O_3 and particles? If there is a common pathway for O_3- and particle-induced toxicity, could it be oxidative stress? Is there any potential for interaction between the two pollutants? If particles produce oxidative stress, what is the mechanism by which this occurs before inflammation makes a contribution?

The decay in toxicity of diesel exhaust particles with time and the observation that aggregates are taken up by epithelial cells (Murphy *et al.,* Section 3.1.1) are important observations since the activation of a mitochondrial driven oxidant cascade within epithelial cells could provide a link between particle pollution and inflammation. The separate examination of the relative toxicities of particles alone and their adsorbed chemicals and any interactions between them should prove informative. Future studies might investigate the role of surfactant, which has been largely ignored.

More research effort should be invested in studies to identify target cells and mechanisms for the effects of O_3 and particles. Current toxicological investigations have limited value in explaining the observed epidemiological findings. It is not clear what mechanisms link the observations arising from toxicological and mechanistic studies, chamber studies and epidemiological studies. It is still the case that no obvious link has been found to explain how particles might affect the cardiovascular system. The protective role of antioxidant pathways, the effect on red cell sequestration and the cytokine-mediated activation of pro-coagulant and plasmolytic pathways are all relevant (e.g. Seaton *et al.,* Section 5.3.4). The marked inflammatory response seen in normal humans exposed to diesel exhaust particles (Nightingale *et al.,* Section 4.1.1; Salvi *et al.,* Section 4.1.2) provides ample scope for multiple cardiovascular pathways to be activated in the microvasculative of the lung. The possibility that small particles pass directly into the circulation is an interesting idea that deserves further consideration. Mediation through the autonomic nervous system may also be a possibility (Tunnicliffe *et al.,* Section 4.2.3)

In future studies, comparisons should be made between effects in susceptible and non-susceptible groups, looking at physiological and chemical end-points as well as biochemical and gene mechanism pathways. An hypothesis is needed to test in what way underlying inflammation may be a predisposing factor; in volunteer studies, investigating susceptible groups seems to be an appropriate way forward.

Different animal models are available to investigate different effects; for example, models of susceptibility may look at pre-existing disease, young *versus* old rats or models of different viral effects. But how relevant are transient events observed in controlled studies in genetically similar experimental animals to the normal human population? In toxicological studies doses can be adjusted to produce the effects to be investigated, but extrapolation from these studies to humans commonly assumes a single-mechanism dose–response curve, which may not be correct or appropriate.

Mechanistic studies should also help to identify dose–response relationships and thresholds.

7.4. Volunteer studies

7.4.1 Role of chamber studies

In chamber studies a number of factors can be investigated; for example, the subjects and the end-points can be varied, interventions can be studied, repeated exposures can be used to allow the development of tolerance, and protective effects, such as the use of antioxidants, can be studied.

Chamber studies have tended to focus on physiological changes. Many such studies have shown airway inflammation and oxidative stress, but it is not clear whether the observations reflect adaptive rather than pathological responses.

In chamber studies, there is often a marked variation in individual response (in lung function, for example) even among 'normal' subjects. In general, with classical air pollutants, apart from SO_2, asthmatics respond no differently to 'normal' subjects, but there may be marked variations within subject groups. Another limitation of chamber studies is that patients with the most severe conditions are normally excluded, yet these are the people most at risk. Thus some sensitive groups of particular interest, such as the elderly, neonates or brittle asthmatics, are not included in such studies.

Chamber studies have a useful role in raising hypotheses, but they are hard to set up, the experiments may be tedious, study groups may be too small in number, and exercise may cause refractoriness and so affect the outcome of the study. Although what the subjects breathe while they are in the chamber is controlled, the results of the study may be affected by the ambient air subjects were breathing before they entered the chamber. The best way to deal with the latter problem is by randomisation, but this requires a larger number of subjects.

Studies in the Joint Research Programmes by Jenkins *et al.* (Section 4.2.1) and Tunnicliffe *et al.* (Section 4.2.2) confirm findings from other studies that air pollutants can enhance asthmatic responses to inhaled allergens. Though the mechanism for such an effect is not clear, a likely explanation is the capacity of irritant pollutants to increase allergen penetration at the physical barrier of the epithelium.

7.4.2 Future directions for research using volunteer studies

Given the limitations of chamber studies, it will be helpful if, in future, they can be more focused, studying greater numbers of people in more specific ways.

Chamber studies are useful in identifying individuals with extreme responses, and in identifying, for example, relevant phenotypes; this cannot be done in time-series studies, for example. In this respect, it might be possible to construct a small panel of people who had taken part in volunteer studies and who had demonstrated a particularly sensitive response, and then to follow them up in an epidemiological study. This would be a substantial undertaking, as it would require a large number of potential panel subjects, who would require biochemical investigation (using a predefined marker of sensitivity) to select individuals for follow up.

7.5 Epidemiology

7.5.1 The contribution of the Joint Research Programmes to research on epidemiology in the UK and internationally

In some of the studies conducted in the Joint Research Programmes the hypotheses being tested have not always been as clear as they might have been. There is a need to focus more on the link between the type of exposure and the observed health effect. Epidemiological studies cannot clearly identify which individual pollutants are causing particular effects, as exposure is always to a complex mixture; yet it is still single pollutants that are regulated. This begs the question as to whether it is possible to find a better way of controlling air pollution than by regulating single pollutants.

A further limitation of studies in the Joint Research Programmes to date is that they have focused on a limited range of pollutants. In particular, PM_{10} has been intensively investigated, but little has been done to examine the pollution mix. Although particulate air pollution is a major priority, other pollutants of concern (particularly for indoor air), include house dust mites, NO_2, carbon monoxide (CO; sublethal effects), and PAHs.

Epidemiological studies that identify the contributions to adverse health effects made by different sources of air pollution, including traffic, (of which there are several in the Joint Research Programmes) are becoming increasingly important. In general, more epidemiological studies could be conducted in the wake of 'natural experiments', such as road by-pass schemes and factory closures.

Time-series studies

Current work on outdoor pollutants is dominated by the time-series studies. These are 'real world' studies, covering the full range of exposures and population sensitivities. The more successful studies on air pollution have been those which have looked at large differences in exposure, which is one of the benefits of time-series studies, where exposure contrasts are provided, for example, by variations in the weather. Theoretically, with a perfect understanding of exposure, effects on health could be predicted.

Although time-series studies can easily identify mortality as an end-point, other important health effects may be missed. Also, time-series studies can provide little information on the importance of peaks and troughs in exposure levels relative to changes in background levels. Furthermore, information on dose–response relationships obtained from time-series studies is usually fairly limited, although it not clear how much the limitations are due to poor classification of exposure. It is also difficult to identify effects in particular subgroups from time-series studies.

Mortality and acute effects

There are several US studies on outdoor air pollution and mortality, but few have been conducted in Europe. Most epidemiological studies conducted within the Joint Research Programmes have similarly focused on the acute effects of short-term and long-term exposures. Daily time-series studies consistently appear to indicate toxicity associated with ambient air that is detectable at a public health level, and evidence that air pollution (especially respirable particles) is associated with acute morbidity and mortality has now been obtained, with considerable consistency in outcome measures, throughout the world (e.g. studies in the Joint Research Programmes by Anderson *et al*., Section 5.4.1). Effects have been most marked in the elderly and those with pre-existing cardiorespiratory disease. As techniques to measure particles improve it will be important to develop epidemiological methodologies that can measure small changes in effect. It may be that little more will be gained from studies on the acute effects of outdoor air pollution, such as the US time-series studies and the APHEA studies, although it is still the case that little is known about the magnitude of the shortening of life that appears to be associated with air pollution. It may be possible to study the phenomenon of 'harvesting', or premature displacement of mortality, by medium-term follow up of susceptible populations (as will be the case in populations in London and Birmingham in the second round of the APHEA study).

More detailed studies on susceptibility (such as those in the Joint Research Programmes by Luczynska *et al.,* Section 5.3.1 and Agius *et al*., Section 5.3.3) should prove informative. The work of Seaton *et al.* (Section 5.3.4) on coagulation factors and the (initially) unexpected increase in cardiovascular morbidity associated with air pollution is also of interest.

Respiratory disorders, asthma and allergy

There are several European cross-sectional studies on respiratory disease, such as SAPALDIA and ISAAC (on asthma), the birth cohort being studied by Strachan *et al.* (Section 5.4.3), and comparisons of bronchitis in eastern and western Germany (e.g. Nicolai, 1997). But there is no evidence to date that air pollution has any impact on the incidence of asthma. Prospective cohort studies, including the several birth cohort studies underway in the UK, (e.g. Bergmann & Woodcock, 1998; Tariq *et al.,* 1998; Atkinson *et al.,* 1999; Burr *et al.,* 1999) are the ideal way to study asthma and to address questions related to both the indoor and outdoor environment. There are also still uncertainties about the links between air pollution and symptoms in patients with chronic obstructive pulmonary disease (COPD).

Some studies in the Joint Research Programmes seem to confirm that outdoor pollutants are able to influence respiratory health in schoolchildren (Ward *et al.,* Section 5.4.2; Venn *et al.,* Section 5.5.1); however, not all studies are positive (Strachan *et al.,* Section 5.5.2).

Although there are some relevant studies in the Joint Research Programmes (by Venn *et al.,* Section 5.1.1; Matti & Clough, Section 5.1.2; Evans *et al.,* Section 5.2.1; Gladding & Crook, Section 5.2.2), more should be done to investigate the importance of biological contaminants in indoor air as determinants of asthma and allergy (identified as an important priority area for research in Europe; see above). There is consistent evidence that particles can interact with other air pollutants to cause adverse effects; inhalation of particles seems to enhance responses to allergens, and NO_2 interacts with bio-allergens. A recent US study (Becker & Soukup, 1999) suggested an interaction between NO_2 and particles to enhance viral infections. Similarly an ongoing UK study (by Holgate and colleagues) investigating the impact of NO_2 exposure among asthmatic children during the virally induced asthma season has demonstrated a good correlation between personal NO_2 levels and exacerbation of symptoms caused by the three most common causes of viral colds.

The role of damp housing on indoor air quality is also an aspect of growing interest. This is important because of the known link between damp housing, overcrowding and poverty. From the study of Evans *et al.* (Section 5.2.1), the temperature of the house emerges as a stronger factor than dampness, but these two will be interrelated. The work of Gladding and Crook (Section 5.2.2) will be especially relevant when trying to explain the effects of humidity on exposure to microbial products.

7.5.2 Future directions for research on health impacts of air pollution

Adverse health effects appear to be seen even at low pollution levels. It is still not understood how such small exposures (especially to particles) can be associated with such dramatic health effects. The effects of (particulate) air pollution seem to be more associated with increments (changes) in pollution levels than actual (average) levels. This suggests that it is not the actual dose *per se* that is important so much as the change in dose and that there are marked differences in susceptibility in the population.

Given the complexity of the situation, it seems appropriate to suggest that the UK should invest its research effort in a few large, multidisciplinary (e.g. exposure monitoring and modelling, mechanisms, toxicology and epidemiology) studies.

Chronic effects

So far, the Joint Research Programmes have concentrated rather extensively on acute effects, even though chronic respiratory effects are likely to be relatively more important. Future development of the

programme should include long-term studies to investigate the association between particulate air pollution and chronic health effects, including long-term cardiovascular disease.

The possibility and practicality of setting up a new UK cohort study or reanalysing data from existing studies to investigate longer-term outcomes (such as shortening of life in terms of months or years rather than a few days) should be considered. However, such data tend to be much less robust than the data on acute effects. Furthermore, it is not clear how effectively cohort studies on air pollution can answer questions about longer-term effects, as there are many confounders associated with the outcomes of interest; numerous US cohort studies have failed to provide definitive answers. Another difficulty with cohort studies, especially with reanalyses of existing cohort data, is the problem of characterising exposure in early life; even exposure *in utero* may be important. To date, attention has focused particularly on PM_{10}, yet few historical data on such exposures are available.

One way forward might be to examine different ways of analysing data from time-series studies by, for example, trying to determine whether the influence of short-term effects can be removed from the analyses in order to use the studies to examine effects in the longer term.

It should also be possible to identify long-term effects in cross-sectional studies, provided confounders can be dealt with adequately and there are enough units of comparison to ensure the power of a study (i.e. 50 rather than six cities would probably be needed).

Susceptible groups

More research is needed to define better what constitutes a susceptible group, and to determine how to identify members of susceptible groups. For example, is lung inflammation a good marker of sensitivity, or is it an adaptive response? There are many wide-ranging factors that may influence susceptibility (including genetic differences). Even for such an extreme example as COPD, there are still no clear data on factors affecting susceptibility; for example, only 20% of smokers suffer severe COPD and it is not known why some individuals and not others are so affected.

Most studies in human populations identify some susceptible groups/individuals. It may be that these are distinct groups, or they may simply reflect individuals at the tails of normal distributions of effect. Cause and effect may, in fact, be interpreted differently by clinicians and others. Short-term studies effectively just measure susceptibility to acute mortality; air pollution is merely one factor among many that may impact upon an individual's health status.

It would be worth revisiting some of the time-series studies, together with detailed experimental studies on biological markers, to see whether it might be possible to use data from the time-series studies to identify causes of death more precisely. It might also be possible to reanalyse some of the time-series studies to see whether susceptible individuals can be identified from data on previous admissions; for example, to investigate whether acute mortality actually occurs in those with pre-existing cardiorespiratory disease. More routine inclusion, in clinical investigations, of factors that might be measures of susceptibility, such as fibrinogen or biomarkers of inflammation, would also have the advantage of making individual studies more directly comparable.

Dietary factors

Diet may well be a more important predictor of health in the UK than air pollution; certainly dietary factors may modify the impacts of air pollution and so any new study on air pollution and health in the UK should probably take dietary factors into account. There are good existing UK studies incorporating nutritional measures and it may be possible to co-ordinate any future air pollution studies with these. However, it is noteworthy that no influence of diet was found in the US six cities studies.

Multidisciplinary approach

A large-scale UK multidisciplinary collaborative effort to investigate defined problems would represent a positive move forward. An important question is whether such an effort should develop new cohorts, use existing cohorts or be based on case–control studies. Whatever decision is made, any such multidisciplinary approach should involve early discussions between those assessing health impact and those measuring exposure, as what is

practically measurable may well influence the study design. Adding on to existing studies is always problematic, as the initial study design will probably not have been set up to accommodate the new questions. It should not be assumed that new cohort studies will be prohibitively expensive; in the long run, especially when value of life considerations are taken in to account, they can represent the best investment of resources.

7.6 Public health impact

7.6.1 The contribution of the Joint Research Programmes to research on public health impact

Consideration should be given to addressing the mismatch between official information and the local experience of air quality, and the perception of vulnerable groups in particular, as found, for example, in one study in the Joint Research Programmes on the public perception of risk (Moffat *et al*., Section 6.1.1). Some differences in perception of air quality may be related to the location of the local air quality monitors. However, current expectations are stretching the monitoring system, which was designed only to cover typical urban situations, to its limit. It should also be recognised that people want to be able to do more to control their own situation when air pollution is high. One area where perception of a problem did not necessarily match toxicological impact was that of smell. Smell is often a major public concern, and people can be perturbed by, for example, the proximity of chemical plants and incinerators, whereas, in fact, most pollution in the vicinity is from traffic. Falling dust and soiling were other concerns, as nuisances, rather than having any health impact.

There does seem to be some confusion among the general public about how to interpret health advice. Although people are not normally advised to stay indoors (except in the case of chemical accidents), advice is sometimes interpreted as meaning this. In the USA, some advice is directed more towards what people are likely to experience, given a particular air quality, and this is seen by some to be more helpful.

7.7 Policy implications and proposals for future work

The purpose of the Joint Research Programmes has been to provide information to facilitate policy development, and it is recognised that it is important to exploit the results coming from the studies in the programmes to do this. However, it is not apparent that sufficient has yet been learned to have major policy impacts. The available information is still fragmented and therefore hard to apply in decision-making. Accepting these limitations, this section reviews some policy areas for which work in the Joint Research Programmes might be relevant, and draws out some of the proposed new research directions, described above, that might be expected to have the most impact on policy in the future.

Some critical tests can be applied to air pollution research to assess its relevance to supporting policy, for example:

- How much does air pollution matter relative to other factors?
- Even if studies do indicate a problem, can anything be done to change the situation? If not, the research may have little relevance to policy.

Future priority areas for policy development, which will benefit from further studies, include levels of pollutants in the environment, adverse health effects that may be associated with such pollutants, and determining what should be regulated and how it should be regulated.

Some current UK research priorities are outlined in the Box.

7.7.1 Levels in the environment

While recognising that it is important to relate exposure monitoring and modelling to biological impact, detailed understanding of cause-effect relationships is not essential in order to set standards to protect health. Much is already done to control air pollution: it may be that any positive benefits from additional control measures will be so small that they will make little difference to the general population. The UK already has policies to control particulate air pollution. On a mass basis, primary particles from diesel vehicle exhaust emissions are to be reduced by 80–90% by 2005; new particle traps will also have the effect of reducing the numbers of particles. A UNECE protocol, adopted in November 1999, on SO_2, NO_x and VOCs will also reduce secondary particles by 50% by 2010. Fiscal measures will encourage the introduction of ultra-low sulphur diesel and gas fuels, for example in lorries and London buses. Also, local air quality management action plans are

Existing research priorities

Current COMEAP research priorities on air pollution include investigations of the following:

- Loss of life expectancy due to acute effects on mortality; are hospital admissions additional or brought forward
- Effects of long-term exposure
- Groups at special risk
- Outcomes other than death and hospital admissions

Other research questions arising from the recent review of UK air quality strategy include the following:

- What is the active component of PM_{10}? (This has important policy implications.)
- How does exposure to NO_2 affect health? (In particular, can any effect be disentangled from that of PM_{10}?)
- How should roadside concentrations be interpreted with respect to health effects? (Peak versus cumulative exposure.)
- Do low levels of CO have an effect on health? (Recent time-series studies indicate an effect.)

required to facilitate pollution control by traffic management.

Studies of exposure and concentrations of pollutants in particular settings no longer have much to offer in policy terms. There is nothing to suggest that any new information arising from studies of indoor air quality is likely to have any impact on policies on outdoor air quality (which are already well advanced). However, continuing research on indoor air quality may be justified on the grounds of increasing understanding about impacts on the individual, and is clearly important to help determine how to protect groups at special risk and identify any necessary policy initiatives/interventions specifically in this area.

In contrast to the situation for outdoor air, there are no policy imperatives to drive research on indoor air pollution. It is not clear that society has much interest or inclination to do anything about air pollution caused by cooking, pets, house dust mites and so on even though these may have more marked impacts on health. Building regulations can be used to influence some indoor sources. But, for example, even if, as is now the case, extractor fans must be installed in all new houses, there is no way to require that these be used. It is important to keep possible interventions in perspective. The need to control sources is recognised, and information on pollution sources is important. Already there is much regulation to control pollution sources in outdoor air but, currently, apart from some building regulations, little is done to control sources of indoor air pollution.

There has, to date, been little examination of the various factors that are determinants of pollution levels; this should be a fundamental step in policy control. The instigation of policy actions to control exposure to particles is dependent on information about which sources give rise to which particles. For example, how important are vehicular emissions relative to atmospheric conditions? The Joint Research Programmes, which have access to wide-ranging expertise, present an ideal way to address such issues.

Improved understanding of how the general population is exposed to air pollutants will contribute to a better understanding of whether there are thresholds for adverse effects, and should, therefore, improve the scientific basis for setting standards. For many pollutants the search for thresholds of effect is illusory and expensive. A better approach would be to define the concentration–effect relationship as well as possible, and then extrapolate downwards, using rigorous cost–benefit criteria to establish standards.

7.7.2 Mechanisms

Although a general relationship between particulate pollution and adverse health impacts is recognised, better understanding of mechanisms of particle toxicity, especially with respect to the physical and chemical characteristics of particles, will improve knowledge about the kinds of emission controls that should be envisaged (e.g. what sources and sizes of particulates to control). In this respect, studies on mechanisms may be more informative than epidemiological studies.

One area where further research on mechanisms might be particularly informative is cardiovascular disease. If air pollution is associated with changes in levels of blood clotting factors, this would provide a clear mechanism by which death could be brought forward by many years.

7.7.3 Adverse health effects

Air pollution is not a major cause of respiratory disease relative to other factors such as smoking. Although acute effects of air pollution are measurable in a large population, it is doubtful that they represent a major public health impact.

Nonetheless, a major policy question is: How much is life shortened by air pollution? The different policy implications associated with a few days of life lost in very ill people versus months or years of life lost are very important. The greater the amount of life lost the more important is the problem, even without considering the societal value of the loss. It is therefore important to seek to quantify what loss of life expectancy is associated with air pollution. However, it should be noted that the major public concern appears to be the *number* of lives that are shortened as an effect of air pollution, rather than the *extent* to which those lives are shortened.

Further acute mortality studies in the UK, looking at the characteristics of people affected on high pollution days, might provide more information on loss of life expectancy; that is: Who is dying on high pollution days? Another way to approach the same question might be to try to determine the number of lives saved on the days when pollution levels fall. Although it may be arguable that COPD patients whose lives are shortened by a few days as a result of air pollution might not have lived much longer anyway, this is possibly not true of those dying of heart disease, the major cause of excess death.

Although it is well accepted that UK air quality is now much improved and there is far less likelihood than in the past that peak exposures will occur that may be damaging to health, there are continuing concerns about the possible adverse impact of long-term low-level air pollution. There are important questions on how much impact such pollution might have in an otherwise healthy environment and how much impact it might have in a disadvantaged environment (e.g. where there is social deprivation). Chronic health effects may have high economic impacts. Future research efforts should concentrate on broader measures of morbidity and relative impacts in healthy and socially deprived environments.

Further studies are therefore needed to understand better the longer-term effects of air pollution. Currently the UK does not use information on long-term effects to determine policy decisions. Nonetheless, it is the costs and impacts of long-term effects of air pollution that appear to drive the public health debate.

It may be arguable that the UK has already reached the point (or can expect to reach the point) at which it is impractical to reduce air pollution levels any further. This might weaken any argument about the cost-effectiveness of initiating any further long-term studies in the UK. As pollution levels are relatively lower than elsewhere, it might be expected that cohort studies in the UK would be much less likely to identify any adverse effect of air pollution than similar studies carried out in Eastern Europe, for example, which is more heavily polluted. However, even in the US six cities studies there have been measurable differences in PM_{10} exposures and, except for a few specific locations, PM_{10} and $PM_{2.5}$ levels in Eastern Europe are not much higher than those in the UK. Long-term studies are better conducted on populations, such as those in the UK, in which there are fewer adverse environmental and socioeconomic influences on health then in many other countries.

At the moment, techniques to measure chronic mortality are not well developed for non-occupational studies. However, it should be possible to transfer expertise (for example from long-term occupational studies on diesel exhaust) from the occupational to the non-occupational field. Some routine surveillance work is already under way (for example studies conducted under the auspices of the WHO Global Environmental Monitoring System) in which data on health are collected from people living near exposure monitoring systems. Periodically the results from the exposure monitoring system can be compared with those from the routine health surveillance to see whether any possible associations can be identified.

There may still be more that can be done to enhance the protection of susceptible groups, however they may be defined (i.e. actually susceptible or simply at the tails of normal distribution curves). Policy decisions based on studies among normal people in normal exposure situations may protect most people very well but, in general, little is known about responses in the vulnerable 10% of the population.

In terms of public health policy it will be important to identify those who are susceptible to serious long-term effects, rather than concentrating on acute effects, as has been done to date. In general, other than for asthmatics, public health actions do not specify susceptible groups or take particular account of them.

7.7.4 Future collaborative programme

Future research in the UK would benefit from a multidisciplinary collaborative approach. The great strength of a collaborative programme of research is that it would build on the outputs and lessons learned from the current Joint Research Programmes and draw together existing expertise from various UK research groups working in epidemiology and atmospheric sciences.

7.8 Summary of proposals for future research

Based on the discussions at the third annual review meeting of the Joint Research Programmes on outdoor and indoor air pollution, a number of areas for future work of particular relevance to the development of UK policy were identified; these are summarised below.

7.8.1 Exposure

- The relevance of the 'personal cloud' of airborne particles and its impact on total personal exposure
- Exposure modelling and its application (e.g. in epidemiology)
- Exposure measurements for source apportionment
- Clarification of dose–response curves, especially important for cost–benefit analysis and policy implications
- Investigating which substances in or on fine particles are important
- Identification of 'standard particles', the use of which could facilitate comparison between experimental studies

7.8.2 Health effects

- Further studies on the degree of life shortening by air pollution episodes
- The relative importance of air pollution and social disadvantage — Is deprivation actually the most potent predictor of hospital admissions?
- The importance of diet as a modifier of response to air pollution
- Investigations of natural experiments (e.g. new road schemes, factory closures)
- Studies of long-term impacts of air pollution

7.8.3 Susceptible groups

- Identifying which sensitivities are important and who are the sensitive individuals
- Investigating marked idiosyncratic effects seen in some individuals in volunteer studies — Are important differences missed by 'measuring the mean'?

7.8.4 Public health impact and policy

- Presentation of air quality information — Smell, visual impact and dust may concern people just as much as the prospect of possible health impacts
- Determining what should be regulated (especially true for particulates)
- Identifying the appropriate policy levers to ensure improvements in public health

7.8.5 A collaborative approach

- Consider establishing a multidisciplinary collaborative project for the next phase of research into air pollution and health effects in the UK, to facilitate a more efficient, less fragmented and less competitive approach

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Annexes

Annex I Research questions forming the basis of the joint DETR/DH/MRC call for proposals on 'Air Pollution and Respiratory Disease': First Joint Research Programme*

Acute effects of exposure to air pollutants

1a Are short-term variations in exposure to outdoor air pollutants in the UK associated with changes in acute morbidity and, if so, what is the magnitude of the effect?

1b Is long-term exposure to air pollution associated with increased incidences of acute respiratory morbidity and mortality and, if so, what is the magnitude of the effect?

2 Which outdoor air pollutants acting either singly, or in combination or together with other factors, are currently responsible for the acute effects of short-term exposures?

3 What are the mechanisms that are involved in the toxicology of the acute effects of short-term exposure to air pollutants acting either singly, or in combination or together with other factors, in humans?

Long-term consequences of exposure to indoor and/or outdoor air pollutants

4 Are short-term and/or long-term exposures to indoor and outdoor air pollutants associated with chronic health effects as characterised by increased morbidity and mortality and, if so, what is the magnitude of the effect?

5 Which indoor and/or outdoor air pollutants acting either singly, or in combination or together with other factors, are responsible for chronic health effects?

6 What are the mechanisms that are involved in the development of chronic health effects associated with exposure to air pollutants acting either singly, or in combination or together with other factors, in humans?

Methodological issues

7 The development of methods for personal sampling and an investigation of the relationship between this and fixed-site monitoring

8 Further investigations to characterise particulate air pollutants currently occurring in the UK

9 Characterisation of exposure–response relationships

10 Characterisation of measures of effect

* Described in full in IEH (1994) *IEH Report on Air Pollution and Respiratory Disease: UK Research Priorities* (Report R2), Leicester, UK, Institute for Environment and Health

Annex II Research questions forming the basis of the joint DETR/DH call for proposals on 'Health Effects of Exposure to Air Pollutants and Damp in the Home': Second Joint Research Programme

A research programme to address specifically air pollution in the home, where those most likely to be affected — small children, the sick and the elderly — spend the majority of their time.

Research to focus on five key areas

- Personal exposure monitoring and modelling
- The relationship between the composition and concentration of air pollutants in the indoor and outdoor environment
- The effects of indoor factors on sensitive individuals and subgroups in the population
- The toxicological interactions between environmental tobacco smoke, allergens and other indoor air pollutants
- The health effects of damp in the home

Particulate matter, carbon monoxide, oxides of nitrogen and organic compounds are the pollutants of special interest.

Annex III Projects funded by the DETR/DH/MRC Joint Research Programmes on Outdoor and Indoor Air Pollution

Relationship between urban pollution and cardiorespiratory ill-health[1]
Dr R Agius, University of Edinburgh
February 1996–February 1998

Use of passive sampling techniques for estimation of exposure to particulates in the non-workplace environment[2]
Dr R Aitken, Institute of Occupational Medicine, Edinburgh
September 1998–August 1999

Effects of air pollution on daily mortality — admissions and general practitioner consultations in London[1]
Prof R Anderson, St George's Hospital Medical School, London
April 1996–December 1999

Sources and chemistry of atmospheric particles[1]
Prof GE Andrews, Department of Fuel and Energy, University of Leeds
December 1994–March 1996

Modelling of indoor and personal exposure to air pollutants[2]
Prof M Ashmore, Imperial College, London/Dept of Environmental Sciences, University of Bradford
January 1998–June 2000

Effects of sulphuric acid and ammonium bisulphate challenge on lung function and response to allergen challenge[1]
Prof J Ayres, Birmingham Heartlands Hospital
April 1996–April 1999

Effects of exposure to motor vehicle traffic at home and school on prevalence, severity and persistence of wheeze in Nottingham school children: A spatial analysis using GIS[1]
Prof J Britton, Nottingham City Hospital
September 1997–September 1998

Effects of relieving traffic congestion on pollutant exposure and respiratory morbidity[1]
Dr M Burr, Centre for Applied Public Health Medicine, Cardiff
February 1996–December 1999

Indoor pollutants as a risk factor for chronic respiratory symptoms in adolescents[1]
Dr J Clough, Dept of Child Health, Southampton General Hospital
February 1996–July 1997

Exposure to indoor air pollution and the risk of upper and lower respiratory disease in asthmatic children and their mothers[2]
Dr J Clough, Dept of Child Health, Southampton General Hospital
March 1998–February 2000

The effect of a combination of pollutants (NO_2 and O_3) at different concentrations and exposure times on the airway response of mild asthmatics to inhaled allergen over a period of 48 hours[1]
Prof RJ Davies, St Bartholomews and the Royal London School of Medicine
July 1996–March 1999

Study of lung function and biochemical and cellular consequences of acute exposure to diesel exhaust in normal and asthmatic subjects[1]
Dr A Frew, Southampton General Hospital
February 1996–January 1998

Consequences for health and effects of damp on the incidence of airborne microbial cell wall components in the home[2]
Dr T Gladding, University of Luton
February 1998–December 2000

Estimation of personal exposure to gaseous and particulate pollutants[1]
Prof RM Harrison, Institute of Public and Environmental Health, University of Birmingham
November 1996–November 1999

Personal exposure monitoring and modelling[2]
Prof RM Harrison, Institute of Public and Environmental Health, University of Birmingham
January 1998–July 2000

Towards assessing and costing the health impacts of ambient particulate air pollution in the UK[1]
Mr JF Hurley, Institute of Occupational Medicine, Edinburgh
August 1996–October 1998

Interaction of respiratory tract lining fluid with ozone: Protective or detrimental to underlying lung cells[1]
Dr F Kelly, St Thomas' Hospital, London
June 1996–May 1997

Secondary school children with asthma[1]
Dr W Keys, National Foundation for Educational Research, Slough
April 1996–July 1997

A case–control study of the effects of common indoor pollutants on the occurrence and severity of chronic childhood wheezing illness in Nottingham school children[2]
Dr S Lewis, Nottingham City Hospital
June 1998–November 1999

Acute effects of particulate air pollution in patients with respiratory disease[1]
Dr C Luczynska, St Thomas' Hospital, London
January 1998–October 1999

Ultrafine environmental particles: Mechanisms of toxicity[1]
Prof W MacNee, Dept of Medicine, University of Edinburgh
January 1997–December 1998

Personal exposure to atmospheric particles[1]
Mr D Mark, Institute of Occupational Health, University of Birmingham
December 1994–March 1996

The effects on health of exposure to air pollutants and damp in the home: Air quality in Solihull West Midlands[2]
Dr S Marks, Gas Research and Technology Centre, Loughborough
January 1998–March 1999

Size-selective personal sampling for indoor allergens[1]
Mr John McAughey, AEA Technology, Abingdon
March 1996–September 1999

Public awareness of air quality and respiratory health: Assessing the impact of health advice[1]
Dr S Moffat, Dept of Epidemiology and Public Health, University of Newcastle upon Tyne
October 1996–March 1999

Effect of traffic generated air pollutants on inflammation in airway disease[1]
Prof A Newman Taylor, Royal Brompton Hospital, London
July 1997–June 1998

Determinants and distribution of personal indoor airborne pollutants in urban populations[2]
Dr M Nieuwenhuijsen, Centre for Environmental Technology, Imperial College, London
June 1998–May 2000

Do particulates from opencast mining impair health?[1]
Dr T Pless-Mulloli, Dept of Epidemiology and Public Health, University of Newcastle upon Tyne
August 1996–February 1999

Mechanisms of ultrafine particle damage to respiratory epithelium[1]
Prof R Richards, School of Biosciences, Cardiff University
May 1996–May 1999

Physicochemical identification of indoor and outdoor particulate matter[2]
Prof R Richards, School of Biosciences, Cardiff University
January 1998–December 2000

Carbon monoxide and nitric oxide exposure in the vulnerable elderly[2]
Dr J Ross, Dept of Environmental and Occupational Medicine, University of Aberdeen
March 1998–February 2000

Air pollution and cardiovascular disease: An investigation of the relationship between particulate air pollution and blood coagulation factors[1]
Prof A Seaton, Dept of Environmental and Occupational Medicine, University of Aberdeen
March 1996–March 1999

An epidemiological study of the impact of damp and other aspects of poor housing on adult health[2]
Dr S Stewart-Brown, Centre for Statistics in Medicine, University of Oxford
April 1998–March 1999

Chronic respiratory health effects of cumulative air pollution exposure: A National Birth Cohort Study[1]
Prof D Strachan, St George's Medical School, London
December 1995–December 1996

Relationship of asthma and allergen rhinitis to local traffic density and ambient pollution modelled at a small area level[1]
Prof D Strachan, St George's Medical School, London
April 1997–March 1998

Comparison of gaseous emissions from different types of pillow[2]
Prof D Strachan, St George's Hospital Medical School, London and Dr D Crump, Building Research Establishment
September 1998–August 1999

Novel exposure system to study antioxidant modulation of effects of air pollutants on lung cells[3]
Dr T Tetley, Charing Cross Hospital, Imperial College, London and Prof. Frank Kelly, St Thomas' Hospital, Kings College, London
August 1999– July 2002

Effects of fine particulate air pollution and acid aerosols on respiratory function and symptoms in school children[1]
Dr S Walters, University of Birmingham Medical School
April 1996–March 1998

Indoor-generated particles and their biological toxicity/Exposure to particulate air pollution and respiratory illness[2]
Dr M Watt, Dept of Environmental and Occupational Medicine, University of Aberdeen
April 1998–March 2000

Distribution of carboxyhaemoglobin levels in the elderly[2]
Dr P Whincup, Royal Free Hospital, London
March 1998–February 2000

[1] Funded under programme on Air Pollution and Respiratory Disease: First Joint Research Programme
[2] Funded under programme on Health Effects of Exposure to Air Pollutants and Damp in the Home: Second Joint Research Programme
[3] MRC Strategic Grant

Annex IV Abstracts presented at the first annual review meeting of the Joint Research Programme on Outdoor and Indoor Air Pollution

Exposure

Personal exposure to benzene and particulate matter
C Thornton, D Mark & RM Harrison

Personal exposure measurements of the general public to atmospheric particles
D Mark, SL Upton, CP Lyons, RA Appleby, EJ Dyment, WD Griffiths & AA Fox

Size-selective sampling of indoor air exposure to tobacco smoke and allergens
J McAughey, I Marshall, C Luczynska & D Mark

The composition of fine particles as a function of size for urban and rural samples
GE Andrews

Volunteer studies

The effect of challenge with fine particles of sulphuric acid and ammonium bisulphate (and sulphur dioxide) in normal and asthmatic adults
WS Tunnicliffe, D Mark, JL Devalia, RJ Davies, RM Harrison & JG Ayres

Acute inflammatory responses in the airways and peripheral blood following short term exposure to diesel exhaust in healthy human volunteers
AJ Frew, SS Salvi, A Blomberg, R Bertil, T Sandström & ST Holgate

The effect of a combination of pollutants (NO_2 + O_3) at different concentration and exposure times on the airway response of mild asthmatics over a period of 48 hours
HS Jenkins, JL Devalia & RJ Davies

Mechanisms

The role of particle size of carbon black in producing oxidant stress and inflammation in rat lung after inhalation
XY Li, K Donaldson & W Macnee

Mechanisms of ultrafine particle damage to respiratory epithelium
RJ Richards, SA Murphy, FD Pooley & K BéruBé

Is the response of the respiratory epithelium to ozone modified by the presence of lung lining fluid?
FJ Kelly & T Tetley

Effect of traffic generated air pollutants on inflammatory airway disease
R Kinnersley

Public impact

Public awareness of air quality and health: Addressing the impact of health advice
S Moffat, C Dunn, J Bush, D Howel & S Young

Risk perception of residents near opencast coal mining sites — a proposal for qualitative research
M Craven, T Pless-Mulloli & S Moffat

Towards assessing and costing the health impacts of ambient particulate air pollution in the UK
JF Hurley, A Markandya, M Holland, HR Anderson, JG Ayres, PT Donnan, RM Harrison, BG Miller, JR Stedman & KG Stevenson

Epidemiological studies

Effects of air pollution on health in London. Part 1: Accident and emergency attendances for respiratory disease
HR Anderson, JM Bland, J Bower, J Emberlin, A Haynes, A McMichael & D Strachan

The effects of relieving traffic congestion on pollutant exposure and respiratory morbidity: the by-pass study
ML Burr, J Layzell & G Karani

Study of the aetiological effect of vehicle traffic pollution on the prevalence of wheeze in Nottinghamshire school children
A Venn, J Britton, S Lewis & M Bell

Chronic respiratory health effects of cumulative air pollution exposure: A national birth cohort study
DP Strachan, K Stevenson & HR Anderson

Urban air pollution and cardiopulmonary ill-health and mortality in Edinburgh
R Agius & G Prescott

Thamesmead respiratory health survey: The acute effects of particulate air pollution in patients with respiratory disease
C Luczynska, J Sterne, P Burney, F Kelly, J McAughey, D Wheeler & J Rice

Does indoor air pollution increase frequency, duration and severity of respiratory symptoms in an adolescent population?
G De Bilderling, A Chauhan, NJ Withers, JL Low, ST Holgate & JB Clough

An investigation of the relationship between particulate airborne pollution and blood coagulation factors
A Seaton, D Godden, J Cherrie, R Stout, V Crawford & R Agius

Does living near opencast coal mining impair health? Design of an environmental epidemiological study
T Pless-Mulloli, D Howel, J Tate & M Craven

The short term health effects of very fine particulate and acid aerosol air pollution upon lung function and respiratory symptoms of asthmatic and non-asthmatic primary schoolchildren: A panel study
DJ Ward, KT Roberts, JG Ayres, S Walters & RM Harrison

Annex V Abstracts presented at the second annual review meeting of the Joint Research Programmes on Outdoor and Indoor Air Pollution

Exposure to particles

Benzene and particulate matter (PM_{10}) in the indoor environment
C Thornton, RM Harrison & D Mark

Determinants and distribution of personal indoor airborne pollutants in urban populations
M Nieuwenhuijsen, P Cullinan & M Ashmore

Measurement of personal exposure to PM_{10} in the non-workplace environment using passive sampling techniques
RJ Aitken & LC Kenny

Size-selective sampling of indoor air exposure to tobacco smoke and allergens
J McAughey, I Marshall, C Luczynska & D Mark

Indoor-outdoor relationships of benzene and particulate matter
N Jones, RM Harrison & D Mark

Exposure to other indoor pollutants

Biological pollutants
C Hunter

Volatile organic compounds in indoor air
D Crump

Indoor air quality — inorganic pollutants
D Ross

Comparison of gaseous emissions from different types of pillow
DP Strachan, D Crump & J Llewellyn

Other studies of exposure

Particulate monitoring and characterisation in relation to opencast mining — preliminary findings
T Pless-Mulloli, D Howel, AM King, IM Stone & JR Merefield

Carbon monoxide and nitric oxide exposure in vulnerable elderly people
JAS Ross & DG Seymour

Physiochemical identification and comparative biopersistence of indoor and outdoor particulate matter
RJ Richards & KA BéruBé

Indoor air quality survey — Solihull, West Midlands
ST Marks

Epidemiological studies

Effects of air pollution on health in London
HR Anderson, JM Bland, J Bower, J Emberlin, A Haynes, A McMichael & D Strachan

The effect of relieving traffic congestion on pollutant exposure and respiratory morbidity — The Bypass Study
M Burr & G Karani

Local traffic flow and the prevalence, severity and persistence of wheeze in schoolchildren: Combined cross-sectional and longitudinal study
A Venn, S Lewis, M Cooper, J Britton, R Boddy & M Bell

Relationship of asthma and allergic rhinitis to local traffic density and ambient pollution modelled at a small area level
D Strachan, P Elliott & D Briggs

The short-term health effects of fine particulate and acid aerosol air pollution upon lung function and respiratory symptoms of asthmatic and non-asthmatic primary school children: A panel study
DJ Ward, KT Roberts, JG Ayres, S Walters & RM Harrison

The acute effects of particulate air pollution in patients with respiratory disease
C Luczynska, M Strangward, F Flood, A Hogg, J Sterne, P Burnley, F Kelly, J McAughey & D Wheeler

Air pollution and cardiovascular disease: An investigation of the relationship between particulate air pollution and blood coagulation factors
A Seaton, D Godden, J Cherrie, R Stout, V Crawford & R Agius

Does indoor air pollution increase frequency, duration and severity of respiratory symptoms in an adolescent population?
G de Bilderling, A Chauhan, NJ Withers, JL Low, ST Holgate & JB Clough

Exposure to indoor air pollution and the risk of upper and lower respiratory disease in asthmatic children and adult women
JB Clough, AJ Chauhan, G de Bilderling & ST Holgate

Relationship between urban air pollution and cardiorespiratory ill-health
G Prescott, GR Cohen, RA Elton, AJ Lee, FGR Fowkes, I Beverland & RM Agius

Epidemiological studies of damp in the home

Consequences for health, and effects of damp on the incidence of airborne microbial cell wall components in the home
T Gladding & B Crook

An epidemiological study of the impact of damp and other aspects of poor housing on adult health
S Stewart-Brown, J Evans, S Hyndman & D Smith

Mechanisms

Indoor generated particles and their biological toxicity
M Watt, A Soutar, J Cherrie, A Seaton & K Donaldson

The effects of four carbon black samples and diesel exhaust particles on isolated type 2 and Clara cells
SAM Murphy, KA BéruBé & RJ Richards

Modelling studies

Modelling of indoor and personal exposures to air pollutants
M Ashmore, M Byrne, S Dimitroulopoulou & R Kinnersley

Personal exposure — monitoring and modelling
RM Harrison, D Mark & JG Ayres

Public health impact

Towards assessing and costing the health impacts of ambient particulate air pollution in the UK
JF Hurley, MR Holland, A Markandya, HR Anderson, JG Ayres, PT Donnan, RM Harrison, K King, BG Miller, JR Stedman & KJ Stevenson

Asthma and air quality: A survey of schools and young people
L Kendall

Public priorities concerning risks to health from air pollution: Work in progress
S Moffatt, C Dunn, J Bush, D Howel & H Prince

Volunteer studies

The effect of challenge with fine particles of sulphuric acid and ammonium bisulphate (and sulphur dioxide) in normal and asthmatic adults
WS Tunnicliffe, D Mark, JL Devalia, RJ Davies, RM Harrison & JG Ayres

Inflammatory effects of diesel exhaust on human airways
A Frew, SS Salvi, A Blomberg, FJ Kelly, TA Sandström & ST Holgate

The effect of a combination of pollutants (NO_2 and O_3) at different concentrations and exposure times on the airway response of mild asthmatics to inhaled allergen over a period of 48 hours
HS Jenkins, RL Mister, JL Devalia & RJ Davies

A development of a method to study the health effects of diesel particulates in humans
R Kinnersley, M Ashmore, P Cullinan, J Nightingale & AJ Newman Taylor

Abstracts not presented

A case–control study of the effects of common indoor pollutants on the occurrence and severity of chronic childhood wheezing illness in Nottingham school children
S Lewis, A Venn & J Britton

Distribution of carboxyhaemoglobin levels in British men aged 60–79 years: The British Regional Heart Study
P Whincup & AP Haines

The role of particle size of carbon black in producing oxidant stress and inflammation in rat lung after inhalation
W Macnee, XY Li & K Donaldson

Completed research projects

The composition of fine particles as a function of size for urban and rural samples
GE Andrews

Personal exposure measurements of the general public to atmospheric particles
D Mark, SL Upton, DP Lyons, RA Appleby, EJ Dyment, WD Griffiths & AA Fox

Interaction of respiratory tract lining fluid with ozone: Protective or detrimental to underlying lung cells
F Kelly & T Tetley

Chronic respiratory health effects of cumulative air pollution exposure: A national birth cohort study
DP Strachan, K Stevenson & HR Anderson

Annex VI Attendees at the third annual review meeting of the Joint Research Programmes on Outdoor and Indoor Air Pollution

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Annex VII Publications arising from the DETR/DH/MRC funded Joint Research Programmes on Outdoor and Indoor Air Pollution*

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Annex VIII Summary conclusions from a meeting to review the first Joint Research Programme on Outdoor and Indoor Air Pollution held at the Institute for Environment and Health on 22 October 1999

Achievements

A major achievement over the six years since the programme's inception is that the UK now has its own air pollution research programme, which is a joint initiative across government departments. The Joint Research Programmes are contributing to increased understanding about the effects of air pollution on human health; particular strengths are in epidemiological, chamber and mechanistic studies. As might be expected, most scientific advances have been incremental, contributing to the body of knowledge about air pollution. A few studies, especially those on mechanisms, have contributed decisive new ideas. The effectiveness of UK research in the field has, it is hoped, been strengthened by the collaborations that the programmes have encouraged both between government departments and agencies and between research teams in the UK. It is also hoped that the success of the programmes has contributed to the increased recognition and influence, both nationally and internationally, being gained by UK researchers in the air pollution field.

Collaboration

- The successful interdepartmental collaboration between DETR, DH and MRC, to promote and fund a joint research exercise has been a remarkable and uncommon achievement.
- The involvement of the MRC in the collaboration with government departments has enhanced the first programme's scientific credibility and independence.

Influence and policy

- The Joint Research Programmes have raised the awareness of problems related to air pollution and, in particular, the importance of indoor air pollution.
- Results from the programmes and increased understanding of air pollution issues in general have provided scientific support for the UK National Air Quality Strategy.
- The programmes provided support to UK Ministers for the setting up and development of the National Air Quality Strategy.
- The priority given to air pollution research in the UK in recent years, the support the Joint Research Programmes have provided to promote UK expertise, and the scientific advances made by the programmes have helped move UK air pollution research to the forefront, both in Europe and elsewhere.
- Increased international recognition of the UK's contribution to air pollution research has strengthened the UK's participation and influence in the EC deliberations on air pollution and in negotiations on EC directives.
- In view of the above points, results from the programmes can be expected, in the future, to have much influence on standard setting at both a national and international level.
- Results from the programmes have improved the knowledge base and so helped both to minimise distortion of knowledge and concern about air pollution by special interest groups and to improve confidence in the credibility of UK experts in the field.

Scientific advances

The Joint Research Programmes, in particular the first programme, have contributed to increased scientific understanding in several key areas and provided answers to some of the important questions on air pollution. A few studies have contributed decisive, new ideas, although most of the advances made by the programmes have been incremental, building on and enhancing existing knowledge. Thus, results from many of the projects in the programme have confirmed findings from studies conducted elsewhere and, in so doing, have strengthened the body of available evidence.

- Projects in the first programme have contributed to a much clearer understanding that acute health effects are associated with short-term variations in air pollution levels, although inconsistencies between studies still have to be addressed. Current opinion considers airborne particles to be the most important pollutant causing adverse health effects in the UK today, together with sulphur dioxide and ozone.

- Key studies in the first programme have drawn attention to the importance of mechanisms and have contributed to a better understanding of the mechanisms of action and toxicology of particles, in particular. Studies in the programme have suggested that particles and ozone may act through similar mechanisms, e.g. by oxidative stress.

Potential developments

While recognising the above achievements of the Joint Research Programmes, including a general positive influence on policy and some particular scientific advances, it is also the case that few specific impacts of the programmes on air pollution policy are clear at present and there are still many unanswered questions. Although the varied nature and breadth of coverage of the projects in the programme render a critical evaluation of the programmes' effectiveness difficult, such an evaluation of the achievements and limitations of the current programme will help determine its cost effectiveness and the future direction of air pollution research in the UK. Some possible ways to maximise the usefulness of the results from the joint programmes and comments on limitations in the programmes are highlighted below.

Policy implications

- The achievements of the programmes should be evaluated to see to what extent they can contribute towards policy decisions that can, in turn, bring about improved health outcomes.
- The economic benefits of changes in policy brought about by the research findings of the programmes should also be assessed; without such a cost–benefit analysis it is difficult to decide whether the programmes have provided value for money.
- It is now possible to weigh the benefits of changes in air pollution policy and the desire to reduce air pollution and, in so doing, to gauge an acceptable cost for any pollution control measure.

Scientific evaluation

- The Committee on the Medical Effects of Air Pollutants (COMEAP) should provide an independent critical evaluation (of as yet undetermined scope) of the contribution to air pollution research of the first joint programme, particularly in epidemiology, mechanisms and chamber studies. Such a review should be set in the context of advances in air pollution research made elsewhere, and should build on a database, currently being compiled, of all published time-series and panel studies on the health impact of air pollution. The review will also need to address apparent inconsistencies in the results of some studies.
- There are too few time-series studies to form a body of evidence from the Joint Research Programme alone; those that have been undertaken would need to be viewed in the context of the overall literature.
- The first programme has provided few data to help elucidate the chronic effects of long-term exposure to low levels of air pollutants.

Directions for future research

Given the wide range of topics that could still benefit from further study, it is important to decide whether a future UK programme on air pollution research should focus on one or two key issues or address a wide range of issues, as has been the case in the Joint Research Programmes to date. With limited funding, it will be important to decide which approach is most likely to result in maximum benefit to the general public. While recognising the importance of research that contributes to the overall body of knowledge, in any future UK air pollution research programme, support should be specifically directed to studies most likely to result in decisive breakthroughs in knowledge and understanding.

Potential key areas for future research continue to focus on the effectiveness of policy interventions, exposure measurement and health effects, as outlined below. Currently attention is directed particularly at particles, although more thought should also be given to the need to continue investigations on nitrogen dioxide.

Continued development of exposure measurement methodology is to be encouraged; inconsistency in the results between some health effects studies may, in part, be explained by poor exposure measurement. Good exposure measurement is also important for the accurate cost–benefit analysis of interventions to change air pollution levels. Health effects research should examine whether any beneficial effect on health impact is gained by interventions that result in differences in air pollution levels that are similar to day to day fluctuations.

Mechanistic studies are one of the most important areas for future research; a better understanding of mechanisms could be expected to influence long-term policy strategies.

More use should be made of natural experiments to investigate air pollution. Such studies may provide information especially directly relevant to cost–benefit analysis. An area of particular debate, which was not resolved by studies in the first joint programme, is that of the effects of road-side pollution.

Policy

- Future research should be more specifically directed towards answering particular policy questions.
- Efforts should be made to investigate whether control measures, by reducing adverse health outcomes, show any cost benefit.

Scientific understanding

Exposure

- Methods for exposure measurement need to be better developed
- In order to establish accurate exposure–response relationships, epidemiological studies require good exposure measurements, in particular of individual exposures.
- The influence of personal behaviour on exposure requires further study. It cannot necessarily be assumed that a reduction in ambient levels will lead to a similar reduction in personal exposure, which may be affected, for example, by differences in activity on high and low pollution days.
- Exposure models could be developed to facilitate cost–benefit analyses.

Health effects

- Future studies should concentrate on investigating the chronic effects of long-term exposure to air pollution.
- The effects of low-level exposures are little understood, and should be addressed in future studies.
- Future epidemiological studies on air pollution should take into account the possible effect of diet on health outcome.
- Time-series studies should be used to investigate whether (and to what extent) air pollution brings deaths forward in time. Studies should investigate extra hospital admissions as well as extra deaths associated with measures of air pollution. Meta-analysis might be a way forward to improve the statistical power of time-series studies.

Particles

- There are still many gaps in the understanding of how exposure to particles, among the most complex of air pollutants, adversely affects human health; further studies are to be encouraged.
- Studies into the kinetics and mechanisms of action of airborne particles should be continued and developed.
- Further studies should be undertaken to investigate the health effects that still apparently continue to arise as a result of exposure to airborne particles.

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Abbreviations

APHEA	Air Pollution and Health: a European Approach
ATD	automated thermal desorber
AU(R)N	automatic urban (and rural) network
BAL	bronchoalveolar lavage (fluid)
Bla g 1	*Blattella germanica* 1
BS	black smoke
CB	carbon black
CBU	cumulative breath unit
COHb	carboxyhaemoglobin
COMEAP	Committee on the Medical Effects of Air Pollutants
COPD	chronic obstructive pulmonary disease
CPC	condensation particle counter
DEP	diesel exhaust particles
Der p 1	*Dermatophagoides pteronyssinus* 1
EPXMA	electron probe X-ray microanalysis
ESD	equivalent spherical diameter
ETS	environmental tobacco smoke
Fel d 1	*Felis domesticus* 1
FESEM	field emission scanning electron microscopy
$FEV_{1\ (0.75)}$	forced expiratory volume in 1 (0.75) second
FID	flame ionisation detector
FVC	forced vital capacity
GGT	γ-glutamyl transpeptidase
GIS	geographical information system
GM-CSF	granulocyte–macrophage colony-stimulating factor
Gro-α	growth-regulated oncogene α
GSH	glutathione
GST	glutathione *S*-transferase
HRV	heart rate variability
IA	image analysis
ICP-MS	inductively coupled plasma — mass spectrometry
IFN-γ	interferon-γ
ILN-1	interleukin 1
ISAAC	International Study of Asthma and Allergies in Childhood
LDL	low density lipoprotein
MCP-1	monocyte chemotactic protein-1
MetHb	methaemoglobin
MI	myocardial infarction
MIP-1α	macrophage inflammatory protein-1α
MMD	mass median diameter
MOUDI	Micro Orifice Uniform Depositing Impactor
NF-κB	nuclear factor-κB
PAH	polycyclic aromatic hydrocarbon
PCV	packed cell volume
$PD_{20\ (15)}$	provocative dose causing 20% (15%) fall in FEV_1
PEF	peak expiratory flow
$PM_{10\ (2.5,\ 1)}$	particulate matter <10 (2.5, 1) µm aerodynamic diameter
PSD-XRD	photosensitive detector — X-ray diffraction
RANTES	regulated upon activation normal T-cell expressed and secreted (cytokine)
ROFA	residual oil fly ash
RTLF	respiratory tract lining fluid
SAPALDIA	Swiss study on Air Pollution and Lung Disease in Adults
SMPS	scanning mobility particle sizer
TAI	traffic activity index
TEAC	Trolox equivalent antioxidant capacity
TEOM	tapered element oscillating microbalance (monitor)
TNF-α	tumour necrosis factor-α
TSP	total suspended particulate (level)
Tunel	TdT-mediated dUTP-digoxygenin nick end-labelling
TWA	time-weighted average
ufCB	ultrafine carbon black
VOC	volatile organic compound

IEH Publications

IEH Reports

Air Pollution and Health: Understanding the Uncertainties	Report R1 (1994)
Air Pollution and Respiratory Disease: UK Research Priorities	Report R2 (1994)
Natural and Man-Made Mineral Fibres: UK Research Priorities	Report R3 (1995)
Perinatal Developmental Neurotoxicity	Report R4 (1996)
The Use of Biomarkers in Environmental Exposure Assessment	Report R5 (1996)
Health Effects of Waste Combustion Products	Report R7 (1997)
Factors Affecting the Absorption of Toxic Metals from the Diet	Report R8 (1998)
Recent UK Blood Lead Surveys	Report R9 (1998)
The Non-auditory Effects of Noise	Report R10 (1997)
Approaches to Predicting Toxicity from Occupational Exposure to Dusts	Report R11 (1999)
Benzene in the Environment: An evaluation of exposure of the UK general population and possible adverse health effects	Report R12 (1999)

IEH Assessments

Environmental Oestrogens: Consequences to Human Health and Wildlife	Assessment A1 (1995)
Indoor Air Quality in the Home: Nitrogen Dioxide, Formaldehyde, Volatile Organic Compounds, House Dust Mites, Fungi and Bacteria	Assessment A2 (1996)
Oilseed Rape: Allergenicity and Irritancy	Assessment A3 (1997)
The Ecological Significance of Endocrine Disruption: Effects on Reproductive Function and Consequences for Natural Populations	Assessment A4 (1999)
Indoor Air Quality in the Home (2): Carbon Monoxide	Assessment A5 (1998)

Special reports

Understanding Asthma (1995)

Health Effects of Ozone and Nitrogen Dioxide in an Integrated Assessment of Air Pollution (1997)

Fibrous Materials in the Environment (1997)

Organophosphorus Esters: An Evaluation of Chronic Neurotoxic Effects (1998)

Joint Research Programmes on Outdoor and Indoor Air Pollution (Review of Progress, 1999) (2000)

Risk Assessment and Toxicology Steering Committee reports

Developing New Approaches to Assessing Risk to Human Health from Chemicals	cr1 (1999)
Risk Assessment Approaches Used by UK Government for Evaluating Human Health Effects from Chemicals	cr2 (1999)
Risk Assessment Strategies in Relation to Population Subgroups	cr3 (1999)
Physiologically-based Pharmacokinetic Modelling: A Potential Tool for use in Risk Assessment	cr4 (1999)
Exposure Assessment in the Evaluation of Risk to Human Health	cr5 (1999)
From Risk Assessment to Risk Management: Dealing with Uncertainty	cr6 (1999)

Food Risk Assessment (FORA)

Probabilistic approaches to food risk assessment	FORA 1 (2000)

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