

# FINE PARTICLES AND HEALTH

- Increased levels of fine particles in urban air are associated with increased mortality.
- Policy implications for the UK.

An earlier POST report<sup>1</sup> looked at the effect of urban air pollution on health, especially asthma, and pointed to emerging evidence that fine particles in the air could be a significant contributor to respiratory disease and death. Since then, there have been many further studies which have reinforced such concerns, and suggest that fine particles from diesels and other sources may contribute to significant mortality around the world.

*This briefing summarises recent developments in this field and examines the policy implications.*

## BACKGROUND

It has long been recognised that air pollution can kill - demonstrated all too vividly in 1952 when 4,000 'extra' deaths were recorded during the 4-day London smog. In those days, smoke particles and sulphur dioxide were the main culprits, and measures followed to clean up the air in cities - initially via smokeless zones, and then through emission controls on power sources, vehicles and industry. As a result, gross effects such as those in the 1950s are a thing of the past. Now the concern is more over the presence of a range of different pollutants - gases such as nitrogen and sulphur oxides combine with hydrocarbons and sunlight to form ozone and give rise to the photochemical 'hazes' on still sunny days in summer and winter.

Even though less serious than in the past, urban air pollution remains a source of respiratory health problems and researchers have been trying to find out more about the effects of the complex mixtures involved on health (see POST report "Breathing in Our Cities"<sup>1</sup>), and which component(s) are most important. As part of this effort, researchers in the USA studied rates of death and respiratory illness to see if they could detect any links between these and day-to-day variations in levels of air pollutants. Early results and a retrospective analysis of data from the London smogs showed a coincidence between fluctuations in levels of **airborne particles** (measured as black smoke) and **variations in death/hospital admission** rates. This preliminary evidence linking fine particles with adverse health effects was considered by the US Environment Protection Agency in the mid-1980s, and led to US air quality standards for fine particles in 1987.

1. Breathing in Our Cities- Urban Air Pollution and Respiratory Health - POST, February 1994.



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Since this time, more sophisticated statistical methods have made it easier to detect so-called 'associations' between relatively small effects on a population's health and possible 'causes', so researchers could study the impact of variations in every-day levels of pollutants rather than just severe episodes. The first such studies in North America showed that even small fluctuations in levels of particulate pollution coincided with changes in mortality rates and hospital admissions.

These findings were initially greeted with considerable scepticism in many quarters, and questions raised over whether they were statistical artifacts, consequences of the limited quality of the data, etc. However, similar associations have now been found in towns and cities throughout North America, Europe (including the first UK study) and elsewhere, and a significant body of evidence has accumulated linking **adverse health effects with levels of airborne particles typical of those encountered in many towns and cities, including those in the UK**. Most attention has been focused on the finer particles which can more readily enter the lung, and thus the debate has tended to be about particulate matter finer than 10 millionths of a metre - microns ( $\mu\text{m}$ ) diameter - otherwise known as PM10. Governments have taken an increasing interest in this matter. In the USA, the 1987 standard is under review. The EU is incorporating new standards into air quality directives. In the UK, this issue has been addressed by the Expert Panel on Air Quality Standards (EPAQS). This POST report looks at what recent research tells us about the sources and effects of airborne particles and the implications for pollution control policy.

**Box 1 WHAT ARE PARTICULATES, HOW ARE THEY CLASSIFIED AND MEASURED?**

In contrast to other air pollutants such as ozone, NO<sub>x</sub> and SO<sub>2</sub>, which are well defined chemical entities, 'particulates' is a generic term describing airborne particles with a wide range of different physical and chemical properties. Their composition thus varies according to geographical location, source, time and the measuring method used, and they may be classified in different ways.

Particulates vary considerably in **size**, from tiny clusters consisting of no more than a few molecules, to larger particles roughly the same size as a fine grain of sand (**Figure 1**). Size determines many of the most important characteristics of a particle, including:

- The time it stays in the air - fine particles (<2.5µm ) remain airborne for days or weeks whereas coarse particles (>2.5µm) fall to earth within a matter of hours.
- The distance it travels - fine particles may travel thousands of kilometres, while coarser ones are deposited closer to their source.
- Whether or not it is measured by a particular detector - e.g. PM10 samplers collect particles smaller than 10µm.
- The extent to which it penetrates the respiratory system - in general, only particles of ~10µm or less are inhaled into the trachea or large airways, and respirable particles (i.e. those that penetrate to the deepest, gas exchanging areas of the lung) need to be even smaller (typically <2.5µm).

Although the exact **physical composition** of particulates will vary from place to place, some generalisations can be made. For instance, the ultra-fine fraction generally accounts for the vast majority of particles in terms of **number**, as illustrated in **Figure 2**. However, because these particles are so small, they account for very little of the overall **mass**, most of which falls in the fine or coarse particle range (Figure 2). Commonly used monitoring techniques such as PM10 are based on measurements of weight, and may thus not be particularly sensitive indicators of the number of ultra-fine particles in the air.

Particles also vary considerably in their **chemical composition**. For instance, in coastal areas particulates will include salt particles from sea spray, whereas in busy urban areas the composition will be much more influenced by road traffic emissions. In practice, most interest has focused on urban air, since most of the UK population live in towns and cities. **Figure 3** illustrates the typical chemical composition of particles monitored in various UK cities, and shows considerable variations with particle size. Overall, the fine (<2.5µm) fraction consists mainly of carbonaceous material (from combustion processes), and of sulphate, nitrate, ammonium and other salts. They thus tend to be acidic and soluble in nature, whereas the larger particles in the coarse fraction (>2.5µm) arise mainly from mechanical processes (grinding, crushing, etc.) and consist largely of insoluble dusts (Figure 3).

Various different terms have been used over the years to classify different fractions of airborne particles - these include:

**Suspended particulate matter (SPM)** are particles that are sufficiently small and light to remain airborne for any significant length of time. In practice, it refers to those <100µm. Based purely on size, SPM is often split into a **coarse fraction** (2.5-100µm) and a **fine fraction** (<2.5µm), with the latter also encompassing another sub-group known as **ultra-fine particles (UFP)**; - <0.2µm). Various different fractions are chosen when it comes to making the actual measurements. Thus **Total suspended particles (TSP)**

refers to the fraction measured by a high volume sampler widely used in the US, and corresponds roughly to diameters up to ~45µm (but varying according to windspeed and orientation). In the UK, the most common measurement has been of **Black Smoke (BS)** where particulate levels are estimated from the blackness of a sampling filter. More recent methods are designed to measure particles in specific size ranges- thus **PM10** and **PM2.5** refer to the samples collected in samplers with 10µm and 2.5µm upper cut offs respectively.

Particulates can also be classified according to the extent to which they penetrate the respiratory system. It is generally thought that the upper cut-off point for particles to be deposited in the windpipe or large airways of the lungs (i.e. **thoracic particles**) is ~10µm, and ~2.5µm and below for those penetrating deeper into the gas-exchanging regions (**respirable particles**).

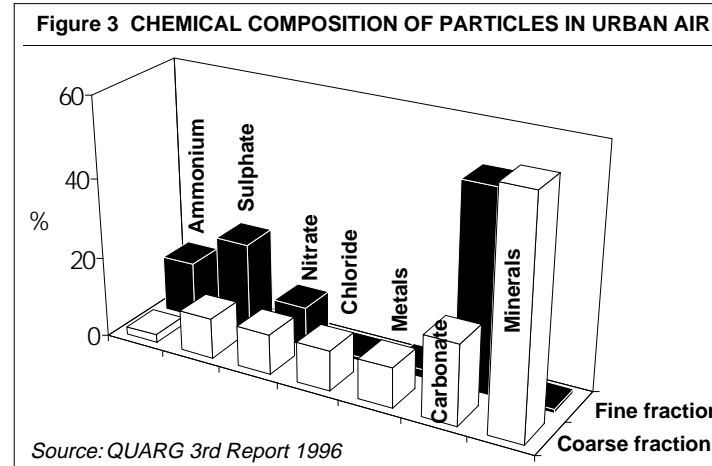
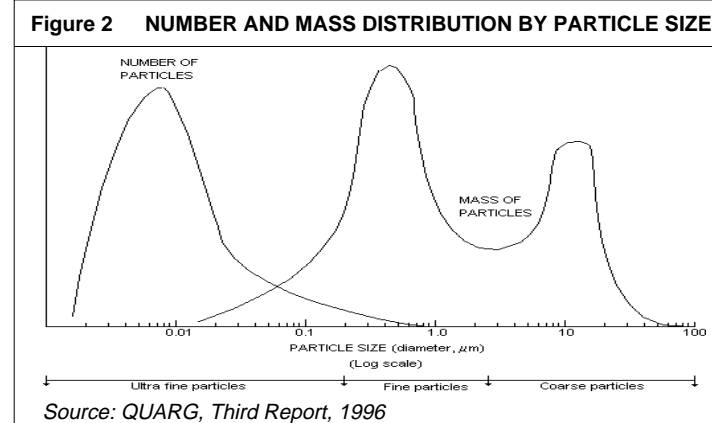
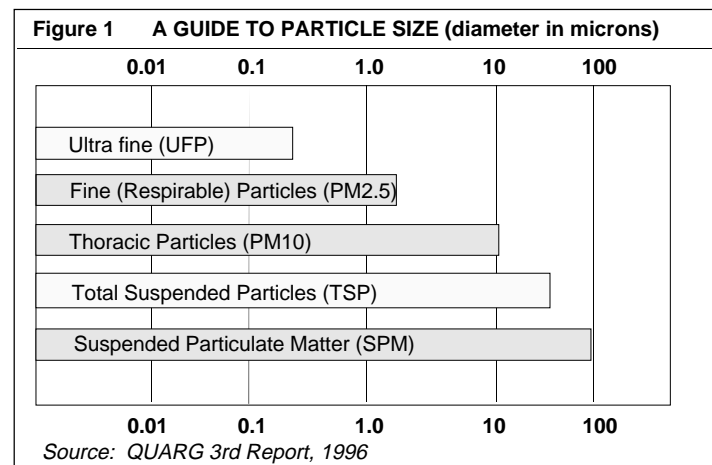
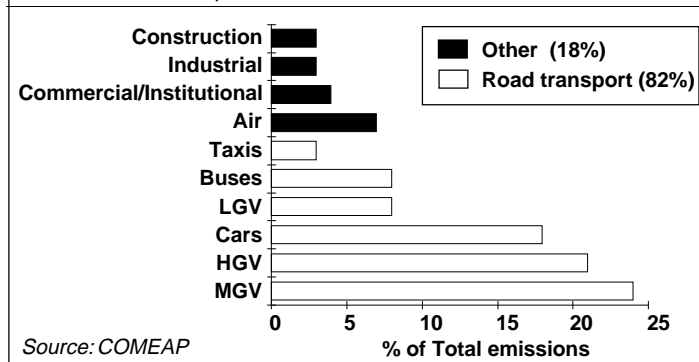


Table 1 NATIONAL INVENTORY OF BLACK SMOKE AND PM10 EMISSIONS (UK, 1993)

Source	% of Total Emission	
	Black smoke	PM10
<b>Combustion sources</b>		
Road transport	51%	25%
Domestic	29%	14%
Waste treatment / disposal	9%	
Power stations	5%	15%
Iron and steel		8%
Refineries	1%	3%
Other industrial combustion	3%	7%
Commercial / public	1%	2%
Other transport	1%	3%
<b>Non-combustion sources</b>		
Industrial		11%
Mining / quarrying		11%
Construction		2%

Source: QUARG 3rd Report, 1996

Figure 4 PM10 EMISSIONS INVENTORY FOR GREATER LONDON, 1993



Source: COMEAP

## SOURCES OF AIRBORNE PARTICLES

Particles in the air we breath come in different sizes and compositions, and are measured in various ways (see **Box 1** on page 2). The variety of the particles themselves is matched by the number of contributing sources, as shown in **Table 1**. Because the old method of measurement (**Box 1**) was based on black smoke (BS), the most recent figures from the National Atmospheric Emissions Inventory (NAEI) have been calculated on this basis, and only recently has attention turned to the finer PM10 fraction. PM10 figures in **Table 1** are thus estimates, since the NAEI's compilers (the National Environmental Technology Centre - NETCEN) are only currently in the process of adding PM10 to the NAEI.

The figures in **Table 1** suggest that road transport is the biggest single source of particulates whether assessed by BS (51% of all emissions) or PM10 (25%). Within this category, diesel emissions are the dominant source, accounting for 48% of BS and 19% of PM10 emissions (and even more in urban areas - see later). Other important sources include domestic combustion (mainly coal), power generation and various industrial combustion processes. In addition, around one quarter of total PM10 emissions are linked to non-combustion sources, the main ones being industrial processes, mining/quarrying and construction.

TABLE 2 UK PM10 LEVELS

SITE	PM10 ( $\mu\text{g}/\text{m}^3$ )		EXCEEDANCES (No days > 50 ( $\mu\text{g}/\text{m}^3$ ))
	Ann.Av	Max.Hourly	
<i>AUN sites (1994)</i>			
Belfast centre	26	490	32
Birmingham centre	23	311	23
Birmingham East	21	319	19
Bristol centre	24	612	30
Cardiff centre	34	564	91
Edinburgh centre	20	307	3
Hull centre	26	264	30
Leeds centre	26	310	44
Leicester centre	21	203	17
Liverpool centre	25	257	34
London Bexley	25	140	18
London Bloomsbury	27	307	39
Newcastle centre	26	297	39
Southampton centre	23	291	16
<i>LAQN sites (1995)</i>			
Bexley	24	351	32
Greenwich	23	253	26
Haringey	26	355	36
Kensington/Chelsea	26	176	25
Tower Hamlets	26	240	37
Thurrock	24	294	17
Westminster	40	397	68

Sources: Air Pollution in the UK, 1994, AEAT; SEIPH.

These inventories do not however provide a complete picture. Aside from the provisional nature of the PM10 estimates, inventories of this kind only take account of **primary** particles - i.e. those emitted pre-formed to the atmosphere. This excludes natural sources (dust, sea spray etc.), as well as most particles in the fine and ultra-fine fractions which are formed in the atmosphere (so-called **secondary** particles) by reactions between various chemicals such as sulphate, nitrate, hydrochloric acid and ammonia, which in turn depend on emissions of other pollutants such as  $\text{SO}_2$  and  $\text{NO}_x$ . Since the majority of all airborne particles in terms of number (**Figure 2 in Box 1**) are the very fine particles, this is an important omission. Another important consideration is that the NAEI is a **national** inventory, and specific locations may well vary considerably from this national 'norm'. For instance, **Figure 4** depicts a PM10 emissions inventory for Greater London, and shows that in this urban area, **road traffic accounts for a much greater proportion (82%)** of total PM10 than is the case across the UK as a whole (25% - see **Table 1**). Again, diesel vehicles appear to be the main sources, with medium (MGV) and heavy goods vehicles (HGV) accounting for nearly 50% of PM10 in Greater London.

## PM10 LEVELS IN THE UK

As described in the earlier POST report, UK monitoring for particles has concentrated mainly on Black Smoke - a legacy of the old 'problems' associated with domestic and industrial use of coal. The growing importance of finer particles has led to a shift in emphasis away from BS and towards measurement of PM10 only relatively recently; a national programme of PM10 measurement came into effect in 1994, with data up to then rather

**Box 2 UK MONITORING OF PM10**

**Detection** - PM10 is monitored by drawing air through a filter with a 10µm cut off point. The particles so filtered out are logged automatically to give the weight (in µg) per volume (m<sup>3</sup>) of air sampled.

**Standards and guidelines** -

- US Environment Protection Agency (EPA) - 24 hour average standard of 150µg/m<sup>3</sup>, annual average standard of 50µg/m<sup>3</sup> (currently under review - new figures have yet to be decided and may focus on smaller particles such as PM 2.5).
- UK - DoE's Expert Panel on Air Quality Standards (EPAQS) recommended a PM10 air quality standard of 50µg/m<sup>3</sup> measured as a 24 hour running average.

**UK monitoring network** - Details of UK PM10 monitoring sites are given in the Table. The first widespread monitoring of PM10 in the UK came with the opening of 12 DoE funded urban sites between 1992 and 1994. Since then a number of other DoE-funded sites have been opened, and the network expanded by integrating Local Authority ('affiliated') sites. The result is the Automated Urban Network (AUN) of 25 sites currently monitoring PM10 and a further 28 expected to come on stream by the end of 1996. Further monitoring is conducted at local authority sites that are not AUN-affiliated - for instance the London Air Quality Network currently has 14 PM10 sites, only 5 of which are affiliated.

**TABLE LOCATION OF DOE-ACCREDITED (AUN) PM10 MONITORING SITES**

Sites currently on stream		Sites expected on stream by end 1996	
Location	Type	Location	Type
Belfast centre	Urban	Belfast East	Urban affiliate
Birmingham centre	Urban	Bolton	Urban affiliate
Birmingham East	Urban affiliate	Bradford centre	Urban
Bristol centre	Urban	Cambridge	Urban affiliate
Cardiff centre	Urban	Coventry centre	Urban
Edinburgh centre	Urban	Glasgow centre	Urban
Hull centre	Urban	Glasgow kerbside	Urban
Kent (Rochester)	Rural affiliate	Leamington Spa	Urban affiliate
Leeds centre	Urban	London Camden	Urban affiliate
Leicester centre	Urban	London Haringey	Urban affiliate
Liverpool centre	Urban	London Hillingdon	Urban
London Bexley	Urban affiliate	London South	Urban
London Bloomsbury	Urban	Londonderry	Urban affiliate
London Eltham	Urban affiliate	Manchester South	Urban
London Kensington	Urban Affiliate	Neath	Urban affiliate
Lough Navar (NI)	Rural	Norwich centre	Urban
Manchester Piccadilly	Urban	Nottingham centre	Urban
Middlesbrough	Urban affiliate	Plymouth centre	Urban
Newcastle centre	Urban	Redcar	Urban affiliate
Sheffield centre	Urban	Salford Bury	Urban affiliate
Southampton centre	Urban	Salford Eccles	Urban affiliate
Sutton roadside	Urban affiliate	Stockport	Urban affiliate
Swansea centre	Urban affiliate	Stoke-on-Trent centre	Urban
Wolverhampton centre	Urban	Thurrock	Urban affiliate

sparse. Current arrangements for monitoring of PM10 are given in **Box 2**.

The original monitoring system was the Department of the Environment's (DoE) enhanced urban network (EUN) under which 12 sites were established in UK cities between 1992 and 1994, monitoring levels of PM10 and other pollutants using automated continuous methods<sup>2</sup>. Since 1994, the PM10 network has expanded rapidly, and by the end of 1996 will consist of an Automated Urban Network (AUN) of 48 sites, the locations of which are given in Box 2. With the exception of two rural sites, all PM10 monitoring will be carried out in urban areas.

Results are available for 14 of the sites listed in Box 2 (1994) and for a further 7 (1995) sites in the London Air Quality Network (LAQN). These are in **Table 2** for:-

- average annual levels, a measure of the overall PM10 background throughout the year;
- maximum hourly values, an indication of the highest level encountered during the year;
- exceedances - the total number of days during the year when the site exceeded the **current recommended UK standard**.

As far as average annual levels of PM10 are concerned, levels are generally in the range 20-30µg/m<sup>3</sup> (see Table 2), which is within the recommended UK standard and that set by other bodies such as the US EPA (Box 2). Westminster (40 µg/m<sup>3</sup>) and Cardiff (34µg/m<sup>3</sup>) had the highest annual average level, although in the case of the latter this was partly due to construction work. Maximum hourly PM10 levels varied considerably from site to site, with the highest value (612µg/m<sup>3</sup>) being recorded in Bristol during a pollution episode in October 1994. All the UK sites operating in 1994 exceeded on many occasions the 50µg/m<sup>3</sup> 24 hour running average standard recommended by EPAQS. Cardiff exceeded the standard for more (91) days than any other site (partly due to the dust from an adjacent construction site), followed by Westminster (68), Leeds (44), London Bloomsbury and Newcastle (both 39 days). The 'cleanest' site was Edinburgh, where the EPAQS standard was only exceeded on 3 days during the year. Because UK monitoring of PM10 is a relatively recent occurrence, no site has been operating long enough to show long-term trends in the levels detected.

2. Other pollutants monitored by EUN sites include SO<sub>2</sub>, NO<sub>2</sub>, CO and ozone (see POST report 'Breathing in Our Cities', February 1994).

## WHAT ARE THE HEALTH EFFECTS?

As already mentioned, a considerable body of evidence has accumulated linking airborne particles with a range of adverse health effects. Most concern acute (i.e. short-term) health effects such as hospital admissions or death, etc., but some also point to long-term exposure to fine particles as a possible factor in a number of chronic conditions. We address these in turn.

### Acute effects

These are typified by the London smog of 1952, when the population was exposed to very high<sup>3</sup> levels of both smoke and SO<sub>2</sub>, and some 4,000 more people died than would normally have been expected for that time of year. The evidence now is that adverse health effects can occur at much lower levels of exposure than on such obviously severe pollution episodes - including those found at current UK monitoring sites.

The evolution of the evidence is described in **Box 3**, based primarily on studies conducted in North America over the last ten years. When all the available data are treated comparably (e.g. by 'ironing out' differences in the particulate measures used), a remarkably consistent picture emerges. Within certain limits (up to PM10 levels of around 200 µg/m<sup>3</sup>), there appears to be a 'straight-line' relationship between dose (i.e. pollution level) and response (e.g. health effects). In general a 10 µg/m<sup>3</sup> rise in PM10 is associated with a 1% increase in mortality, irrespective of whether the rise is from (say) 25-35 or 60-70 µg/m<sup>3</sup>. Whether or not this linear relationship holds true at very low levels of pollution (say below 20 µg/m<sup>3</sup>) is not known, although the models currently do not suggest there is a threshold below which adverse effects cease. Other effects are also revealed - again, for each 10 µg/m<sup>3</sup> rise in PM10 **hospital admissions** for asthma rise by ~2% and by just under 1% for all respiratory conditions. Exacerbation of asthma and a very small decrease in measured lung function are also seen, although the magnitude of these effects varies more from study to study (see Box 3).

The primary studies were largely confined to North America and mainland Europe, and until recently, no information was available to assess whether similar links were found in the UK. This situation has now changed, since the results of a DoE-funded study looking at PM10 levels and health indicators in Birmingham have recently become available<sup>4</sup>. These are summarised in **Table 3**, and are consistent with the results from studies elsewhere (described in Box 3). **It thus appears that the links between PM10 and health indicators**

3. Average daily levels for SO<sub>2</sub> were ~1,340 ppb, and ~4,460 µg/m<sup>3</sup> for smoke.

4. These results were available to EPAQS when this panel considered their recommendation for a PM10 standard in 1995, and are expected to be published by summer 1996.

TABLE 3 PM10, MORTALITY RATES AND HOSPITAL ADMISSIONS - A UK STUDY

Health effect	% daily increase per 10 µg/m <sup>3</sup> rise in PM10
<b>Mortality</b>	
All causes#	1.1%
COPD deaths*	5.0%
Circulatory deaths#	1.7%
<b>Hospital Admissions</b>	
All respiratory*	2.4%
Asthma+	3.3%
Bronchitis*	5.8%
Pneumonia~	5.7%
Cerebrovascular*	2.1%

Notes: COPD (chronic obstructive pulmonary disease); \*same day; #1 day lag; +2 day lag; ~3 day lag.

Source: Prof J Ayres, Heartlands Hospital, Birmingham.

**such as mortality rates and hospital admissions first identified in the USA also occur in the UK, despite differences in climate, sources of PM10, etc.**

Taken together these studies provide strong evidence of a statistically significant link between levels of airborne particles and excess deaths and illness, but do not prove that they actually **cause** these effects. Such proof would require an understanding of the mechanism involved, to explain how relatively low concentrations of particles could exert such apparent effects, and here, the scientific work is still evolving.

One of the main problems is that 'particulates' are such a 'mixed bag' - even a specific measure such as PM10 will include particles of various shapes and sizes with widely differing chemical characteristics. As detailed epidemiological studies look into the effects of different fractions however, it appears that the statistical significance of the links observed increases as particle size decreases - in one study the health effects were most closely correlated with levels of the very fine sulphate particles, followed by PM2.5, PM10, and least well with total suspended particles (TSP).

This has placed the spotlight on ultra-fine particles (UFPs) as the fraction of most interest from the point of view of health effects. A number of factors support this view - for instance, UFPs are deposited in the deepest (gas exchanging) regions of the lung, are the most numerous airborne particles, and account for the vast majority of particulate surface area (see below). There are biologically plausible mechanisms to explain how UFPs could cause illness and death, but experts disagree as to what is the key characteristic of a particle with respect to causing such effects. Possibilities include one or more of the following:

- **size / number** - UFPs could exert their effects simply by virtue of being small enough to penetrate the deeper regions of the lung. One theory is that UFPs cause inflammation, exacerbating lung disease and causing cardiovascular deaths by increasing the coagulability of the blood. Evidence for this comes from the fact that UFPs cause greater inflammation

## Box 3 POPULATION STUDIES OF PARTICULATES AND ACUTE HEALTH EFFECTS

Evidence linking particulates with adverse acute health effects come from 2 types of population study:

- **time series studies**, where large populations (e.g. of towns or cities) are followed over a period of time (months or years) and information gathered on various health indicators (e.g. mortality, hospital admissions) and levels of particulates (measured as BS, PM10, PM2.5, sulphate, etc.). Because such studies are conducted at the population level, individual risk factors such as smoking, socio-economic status, etc. can be assumed to remain constant, and thus not influence the results of the study. Other confounding factors that influence the whole population (e.g. flu epidemics, weather, levels of other pollutants) have to be monitored and taken into account. Complex statistical techniques are then used to see whether day-to-day trends in health indicators coincide with those in levels of particulates, and to assess the significance of any such links;
- **panel studies** are conducted using smaller, specially selected (e.g. schoolchildren, asthmatics), populations (i.e. panels), which are followed for shorter periods of time (weeks or months) to see whether their reported health indicators (e.g. asthma attacks) coincide with fluctuations in particulate levels. Because this approach operates at the individual level, the statistical analysis used must take into account variations in individual factors such as smoking, socio-economic status, etc. that could also influence the health indicator studied.

A number of studies using these approaches were conducted in the USA and elsewhere during the 1980s and '90s, but because various different measures of particulates (BS, PM10, PM2.5, etc) were used, they did not yield results that were readily comparable with each other. However, recent "meta-analyses" have

SUMMARY OF RESULTS FROM MAIN STUDIES		
Health Effect	Study type (number)	% change in health effect per 10µg/m <sup>3</sup> rise in PM10 (range of study means)
<b>Mortality</b>		
Total mortality	Epidemiological (8)	1.0% (0.7 - 1.6%)
Respiratory mortality	Epidemiological (4)	3.4% (1.5 - 3.7%)
Cardiovascular mortality	Epidemiological (4)	1.4% (0.8 - 1.8%)
<b>Hospital admissions</b>		
Asthma	Epidemiological (3)	1.9% (1.9 - 2.1%)
All respiratory conditions	Epidemiological (4)	0.8% (0.8 - 3.4%)
<b>Exacerbation of asthma</b>		
Bronchodilator use	Panel (3)	2.9% (2.3 - 12.0%)
Asthma attacks	Panel (3)	3.0% (1.1 - 11.5%)
<b>Measures of lung function</b>		
Forced expired volume	Panel (4)	0.15% (0.05 - 0.35%)
Peak expiratory flow	Panel (6)	0.08% (0.04 - 0.25%)

Source: Dockery and Pope, 1994.

gone back over these studies and 'translated' the results into a standard metric using conversion factors. A summary of one such analysis, where the results have all been expressed in terms of PM10 (in this case the % change in health effect observed when PM10 levels rise by 10 µg/m<sup>3</sup>) is shown in the **Table**. Overall, the studies show that:

- **statistically significant links** exist between particulates and short-term adverse health effects;
- there is no evidence of any **threshold** below which such effects do not occur - at relatively low levels (say <200µg/m<sup>3</sup>) models suggest that the exposure / response curve is essentially a straight line;
- the results for **mortality** and **hospital admissions** are consistent, with studies carried out in different cities at different times all yielding very similar results - e.g. all 8 studies looking at total mortality found an increase of around 1% (range 0.7% to 1.6%) per 10µg/m<sup>3</sup> rise in PM10;
- results for other health indicators were more variable - e.g. the average rise in exacerbation of **asthma symptoms** was around 3% for both health indicators investigated, but in each case the range was quite wide (2.3%-12.0% for bronchodilator use and 1.1%-11.5% for asthma attacks). Much smaller effects were seen for measures of **lung function** (see Table).

than larger particles of the same substance, and from animal studies where rats exposed to UFPs made of chemically unreactive material (PTFE) suffered from acute inflammation of the lung, and died within 30 minutes.

- **chemistry** - others suggest that the chemical composition of UFPs may be more important, with damage caused primarily by acidic particles. As noted previously, there is some epidemiological evidence to support this.
- **surface area** - because of their small size and large number, UFPs represent an enormous surface area. Some have suggested that damage may result from reactive species (e.g. traces of metals such as vanadium, iron and nickel) trapped on their surface.

Overall, studies on the short-term health effects of particulates raise a number of important questions. For instance, can we trust the complex statistical analyses

on which they are based? If so, what are the public health implications for exposure to the levels of PM10 encountered in the UK? What are the implications for air quality standards and abatement policies? These and other issues are discussed in more detail later.

### Chronic effects

Although studies on acute health effects have taken centre stage in recent years, there are also concerns over the potential impact of longer-term exposure to airborne particles. The research needed here is particularly difficult, since very large populations are required to allow small effects to be detected at a statistically significant level. Factors such as smoking, social/economic status and occupation also have to be taken into account at the individual level. Many early studies comparing death and illness rates at different levels of particulate pollution failed to take account of these potentially confounding factors.

More reliable evidence on the long-term effects comes from two recent studies in the USA. One followed over 8,000 adults living in 6 US cities for a period of 14 years, collecting information from each on factors such as smoking, occupation, etc. Once these had been taken into account, the study found that the difference in pollution levels between the most- and least-polluted city (~18.6 $\mu\text{g}/\text{m}^3$  PM<sub>2.5</sub>) was associated with a 26% increase in mortality. Similar results were found in the second study, which involved more than half a million people in 151 US cities over a period of 7 years, and found that the difference in pollution between the least- and most-polluted city (in this case 24.5 $\mu\text{g}/\text{m}^3$  PM<sub>2.5</sub>) was associated with a 17% increase in mortality.

Both these studies found the increases in mortality to be largely attributable to circulatory and respiratory causes. There were also indications of links between particulate levels and deaths from lung cancer, although these were not always statistically significant.

There is also evidence linking exposure to diesel exhaust with higher rates of lung cancer, based on animal experiments and epidemiological investigations of people whose occupation exposes them to high levels of such fumes (e.g. diesel locomotive maintenance engineers in the USA). However, the high levels of exposure involved makes extrapolation to every-day levels difficult, and the DH's Committee on the Medical Effects of Air Pollutants (COMEAP) concluded that the risk of lung cancer from concentrations of diesel smoke found in UK streets is "exceedingly small".

## ISSUES

### *How Reliable is the Evidence?*

Concerns over the health effects of small particles hinge largely on epidemiological evidence (described in Box 3). These studies have been questioned on a number of grounds. For instance, it has been suggested that the health effects observed were not the result of particulate pollution at all, but rather were:

- artefacts of the statistical methods used;
- the result of flaws in study design;
- caused by other air pollutants;
- caused by other factors (e.g. weather).

Such criticisms have prompted close scrutiny of the studies by a number of expert bodies including COMEAP, WHO, the European Commission, US EPA and the US Health Effects Institute (HEI). These bodies have given the statistical techniques used a clean bill of health - for instance, the DH's COMEAP concluded when it examined the available evidence in 1995 that "the statistical methods used in the principal papers are reliable and appropriate" and a detailed replication and validation exercise using original data from 6 of the

studies conducted by the US HEI found that "it is reasonable to conclude that, in these six data sets, daily mortality ....increased as levels of particulate air pollution indexes increased".

Other questions concerning **study design** include concerns over factors such as the reliability of the measures used (e.g. of health effects and of air pollution levels) and the assumptions used in statistical techniques of meta-analysis to convert measurements on one basis (e.g. black smoke) to the common metric of PM<sub>10</sub>. Again, the studies survive scrutiny by expert groups; e.g. COMEAP conclude that "it appears that there are no major and fundamental flaws in study design" or "in reliability of measures of health effects and of exposure".

When it comes to the possibility of effects being due to **other air pollutants** such as SO<sub>2</sub>, NO<sub>x</sub>, and ozone, the situation is less clear-cut. A number of the studies described previously have looked at these pollutants, and found evidence that some of them (notably SO<sub>2</sub>) may contribute to the observed effects on health, as described in the earlier POST report. Detailed re-analysis of one of the US studies (in Philadelphia) led the US HEI to call for "caution in assuming that this association (between particles and health) represents an independent effect of particles alone". However, this does not invalidate the view that day to day fluctuations in particulates cause mortality and illness - it merely shows that in this particular case mortality could not be attributed **solely** to particles. COMEAP have considered the possible role of other air pollutants and concluded that "much the strongest and most consistent evidence of acute effects is that implicating PM<sub>10</sub>" and that "the evidence supports a view that the role of SPM (particulates) is more fundamental than that of SO<sub>2</sub>".

Finally, there is the possibility that the observed effects could be due to some **other factor** (or factors) which has been overlooked (or not taken fully into account) by the investigators. Factors such as infectious diseases (e.g. flu epidemics) and variations in the weather are the prime candidates here, since these can cause illness and death on a much bigger scale than air pollution. Weather effects are known to be particularly important, and although the studies in question did allow for these, the models used to assess their magnitude were relatively crude, relying on simple daily measures of temperature. Critics point out that there is more to weather than just temperature and that factors such as air pressure and speed, humidity, etc. may also have an important bearing on rates of illness and death. COMEAP considered this question and suggested that the consistency of the results taken as a whole, from studies representing a wide variation in weather conditions, gives "reassurance that the apparent effect of air pollution on mortality is not an artefact of incomplete adjustment for weather".

Overall then, the epidemiological evidence has withstood intensive academic scrutiny and emerged relatively unscathed. Some room for doubt has emerged as to whether particulates alone can account for all the effects observed in all the studies. But despite this, the consistence and coherence of studies conducted in different cities with different climates and pollution mixes has led to a general consensus among experts that the observed effects on mortality are real. As pointed out earlier, final proof that exposure to relatively low levels of particulates actually **cause** death and illness will not be forthcoming until the mechanism(s) involved are known, but in the meantime, COMEAP concluded that **there are strong indications of a causal link between fine airborne particles and effects on health.**

### ***Number of people affected***

Of course, any effects on health due to elevated levels of particulate pollution are superimposed on the normal daily toll of respiratory disease. The WHO Working Group on Air Pollutants estimates that for a 'standard' population of 1 million people, one would normally expect to see some 80 deaths and 60 hospital admissions for respiratory conditions over an average 3 day period. If the same population is exposed to an average PM10 concentration of  $50\mu\text{g}/\text{m}^3$  for three days, it calculates that this would result in 4 deaths and 6 hospital admissions above the norm - increases of 5% in the number of deaths and of 10% in illness.

To what extent are such PM10 levels currently encountered by people living in towns and cities in the UK? As summarised in Table 2, urban monitoring shows that every site operating in the UK exceeded the EPAQS recommended  $50\mu\text{g}/\text{m}^3$  24 hour average standard on at least 3 days during 1994, with peak (hourly) levels ranging between  $140\text{--}612\mu\text{g}/\text{m}^3$ . The London Air Quality Network's data for 1994 suggests that the population of London (some 4 million people in the inner city alone) were exposed to PM10 levels far higher than  $50\mu\text{g}/\text{m}^3$  for a period of 1 or more days<sup>5</sup> on 3 separate occasions during this year. If the WHO estimates are correct (and applicable to the UK - see later), it appears that levels of PM10 currently encountered in towns and cities throughout the UK are sufficiently high to cause increased deaths and hospital admissions on several occasions each year.

But how many people may be involved? The detailed study of mortality in Birmingham concluded that, if the EPAQS recommended standard was always met, there would be 62 fewer deaths per million population each year (relative to Birmingham's typical levels of PM10). Assuming the Birmingham model applies to inner London, which has a population of around 4 million

people, somewhat in excess of 250 fewer deaths each year might be expected if the levels were kept below  $50\mu\text{g}/\text{m}^3$ . Indeed, PM10 levels are considerably higher than in Birmingham<sup>6</sup>, so it is possible the benefits would be significantly in excess of this number.

Some have also attempted to estimate the number of deaths across the whole country which might be attributable to airborne particles, by applying the '10 $\mu\text{g}/\text{m}^3$  of PM10 = a 1% increase in mortality' rule to the whole population. Since, there are around 645,000 deaths in the UK each year, 68% of which (438,600) occur in urban areas, and the weighted average annual PM10 levels in UK urban areas is around  $25\mu\text{g}/\text{m}^3$ , a crude calculation suggests that PM10 could be linked to some 10,965 (25% of 438,600) deaths in urban areas each year.

Such figures have appeared in the media, but have to be treated with considerable caution for a number of reasons. First, the calculation assumes that the 'dose response' relationship is linear between 0 and  $25\mu\text{g}/\text{m}^3$  which, as discussed earlier, is as yet undemonstrated. Secondly, to see such figures as indicating the scale of avoidable mortality may be misleading, since even the most pristine environment will have PM10 levels above zero. Perhaps a safer way of expressing the 'numbers' is to say that at levels currently encountered, each  $1\mu\text{g}/\text{m}^3$  of PM10 is equivalent to ~400 lives or deaths nationwide. In other words, if measures were taken to reduce average annual levels of PM10 (as recommended by EPAQS), each  $1\mu\text{g}/\text{m}^3$  reduction might reduce the number of deaths each year by 400 or so.

Finally, these statistics say nothing of how soon those affected would have died anyway (see next Section), and there are also questions over whether it is valid to extrapolate national figures from monitoring data covering only ~1 in 7 of the UK population. **Such uncertainties mean that all figures are only indicative of the possible scale of effect and not predictions; they are best viewed with a considerable degree of caution. As COMEAP has pointed out, current estimates are likely to provide "only a first approximation to the actual effect" and "studies should be undertaken urgently to allow better quantitative predictions to be made".**

### ***Who is most affected?***

Statistics may allow us to anticipate an increase in the number of deaths when particulate levels are high, but they do not tell us anything about which individuals are affected, or their life expectancy in the absence of elevated particulate levels. One effect of particles could well be to cause those on the brink of death to die a few

5. The 3 pollution episodes in London during 1994 occurred on July 9th-13th, October 14th and December 23rd-25th.

6. The number of days when the 24hr average exceeded 50 ranged from 36-68 in inner London, compared with Birmingham's 19-23.



days or weeks early (so-called 'harvesting'). On the other hand, if airborne particles increase mortality in otherwise healthy people with a life expectancy measured in years, then the public health implications would be quite different.

Determining which of these is the dominant effect is not straightforward, as studies in this area are particularly difficult to design. The limited evidence available suggests that:

- older people (65 plus) are at a higher risk of death on high pollution days than other age groups;
- the excess deaths appear to be mainly from respiratory/ circulatory problems;
- long-term differences in mortality rates linked to pollution levels have been observed.

The first two of these points suggest that particulates often act as 'the straw that breaks the camel's back' - i.e. by exacerbating the severity of acute events (pneumonia, heart attack) in people whose health is already compromised by disease and/or ageing. On the face of it, these findings appear to support the 'harvesting' theory. However, if airborne particles are only bringing forward death by a matter of days or weeks, then from year to year, the fluctuations should level out and no long-term differences in mortality (e.g. between populations exposed to different levels of pollution) would be expected. The fact that such long-term differences are observed suggests that (at least in some cases) **particulates are affecting individuals who might otherwise have lived for some years.**

Overall then, the somewhat patchy and inconclusive evidence available suggests that particulate-related mortality is not solely a question of 'harvesting' the most vulnerable members of the population. Although this may be the dominant effect, longitudinal studies in the USA suggest that sufficient years of life are lost to have a detectable impact on long-term mortality rates. Estimates of the average length of life lost vary considerably, with the most recent (based on as yet unpublished statistical analyses of US longitudinal studies) putting the average figure at around 2 years.

### **Standards and monitoring**

PM10 was originally adopted as the most relevant particulate measure partly because of epidemiological studies in the 1980s linking it with short-term fluctuations in rates of illness and death, and partly because 10µm was seen as an obvious cut-off point for particles able to penetrate into the lung. But more recent studies suggest that it is the smaller particles still which have the most damaging effect, since they are most likely to be deposited in the deepest regions of the lung. Attention is thus turning to the fine (i.e. PM2.5) and ultra-fine fractions as being of most concern.

Such studies raise the question as to whether PM10 is really the most appropriate particulate measure from the point of view of setting air quality standards and the monitoring which assesses compliance. Much consideration has been given to this issue in the USA, where the EPA is expected shortly to announce that it is **setting new air quality standards for PM2.5 to augment or replace existing figures for PM10.** One of the options under consideration is to tighten the existing 24 hour standard (150) to a value closer to the UK EPAQS recommendation (50), but to express this as PM2.5 rather than PM10. The main reasons for selecting PM2.5 are that:

- it is seen as a more biologically relevant indicator;
- PM2.5 is the driving force behind (and the predominant fraction of) the biggest rises in PM10 levels in urban areas (especially in winter);
- sources of PM2.5 are well defined and controllable, whereas those of PM10 are more diffuse and difficult to control;
- levels of PM2.5 measured at fixed sites may give a more accurate picture of personal exposures (because fine particles are more evenly distributed, have longer lifetimes, are found indoors as well as outdoors, etc.).

However, this new focus on PM2.5 does not mean that it is considered to be the **cause** of adverse health effects, merely that it is currently regarded as a more appropriate indicator than PM10. Indeed, experts point out that PM2.5 may be no more than a proxy for some other particulate sub-fraction (or fractions) that will eventually prove to be the most important cause of the observed health effects. Opinion is divided as to what the most likely characteristics of that fraction will prove to be. Some have suggested that acidic particles are most important, in which case measures of sulphate or acid aerosol would be most appropriate. Others believe that it is the number (or total surface area) of inhaled particles that is the key, in which case a count of UFPs may prove to be the most appropriate measure.

In the UK, PM10 remains the focus both for monitoring and air quality standards. As mentioned above, the number of DoE-accredited monitoring sites will have expanded to 48 by end 1996. As for standards, EPAQS recommended in 1995 that measures should be taken to reduce people's exposure to both 'background' (by reducing annual average PM10 levels) and 'peak' (by recommending a 24 hour rolling average standard) PM10 levels. These EPAQS recommendations have yet to be formally accepted by the Government, and policy in this area must also take into account the proposed EU Directive on Air Quality and Management. This will come before the Council of Ministers in June 1996, and when introduced will lead to revised air quality standards under a series of 'daughter' directives.

Two options for policy-makers are thus:

- whether to adopt PM<sub>2.5</sub> as an interim metric for standards and monitoring;
- or whether to stick with PM<sub>10</sub> (e.g. by implementing the EPAQS recommendations) and wait until more is known about the particulate fraction responsible for adverse health effects before considering the use of another measure.

This matter has been recently considered by both the DoE's Quality of Urban Air Review Group (QUARG) and EPAQS. For instance, QUARG made a number of research recommendations in its most recent report concerning particulate monitoring, and these included:

- monitoring PM<sub>2.5</sub> at a number of urban sites;
- measuring particle number counts on an experimental basis at urban, rural and roadside sites;
- studies on particle surface chemical composition;
- studies of the spatial distribution of PM<sub>10</sub> and PM<sub>2.5</sub> across urban areas.

Such measures should help to resolve some of the uncertainties over how best to assess particle levels from the point of view of health impacts. In the meantime EPAQS is keeping the situation under review and, in setting the 1995 PM<sub>10</sub> standard, EPAQS noted that *"it is possible that advances in understanding may in the future indicate that another measure which excludes the larger particles (e.g. PM<sub>2.5</sub>) may be more appropriate"*.

### Control policies

Monitoring results for PM<sub>10</sub> presented previously show that the number of days on which the EPAQS 24 hour standard was exceeded during 1994 varied between 3 (Edinburgh) and 91 (Cardiff), and it therefore appears that there is some way to go before compliance with the new EPAQS recommendation is routine, and even greater challenges if the standard were to be tightened.

The question as to what to control depends on the main **sources** of particulates, and these are likely to vary from one location to another. Despite this, emissions inventories and monitoring studies have allowed QUARG to make a number of generalisations about the sources (and thus the main targets for control) of particulates in urban areas:

- most of the PM<sub>10</sub> fraction measured during pollution episodes actually consists of PM<sub>2.5</sub> (especially in winter);
- virtually all of this PM<sub>2.5</sub> is either directly emitted (**primary particles**) from vehicle exhausts, or formed indirectly (**secondary particles**) by reaction between emissions (NO<sub>x</sub>, SO<sub>2</sub>, etc.) from easily identifiable sources (e.g. vehicle exhausts, power stations and domestic fuel);
- the relative importance of primary and secondary particles varies with the season - in summer, sec-

ondary particles are the main cause of particulate pollution, whereas in winter it is primary particles from vehicle exhausts.

These considerations led QUARG to conclude that control policies should focus on PM<sub>2.5</sub> emissions and would need to taken into account both primary (from vehicle exhausts) and secondary particles (from vehicle exhaust emissions and fixed combustion sources such as power stations). Recent years have seen a number of policies designed to reduce emissions from both these categories of sources, and the likely impact of these policies on future levels of particulates has to be taken into account before assessing whether further controls are required.

Sophisticated mathematical models have been developed to allow the future impact of current policies to be estimated. As far as secondary particles are concerned, the most relevant recent policies are the:

- Oslo Protocol to the UNECE international convention on Long Range Transboundary Air Pollution (which should lead to ~60% reduction in SO<sub>2</sub> emissions across Europe by 2010);
- Sofia Protocol to the UNECE convention (which should freeze NO<sub>x</sub> emissions at 1987 levels);
- EU Large Combustion Plant Directive (which should decrease NO<sub>x</sub> emissions from this source);
- various EU Directives requiring installation of 3-way catalysts on petrol-engined motor vehicles and reducing emissions from diesel vehicles.

Taken together, the impact of these policies is expected to lead to an overall reduction in secondary PM<sub>10</sub> particulate levels of around 39% by the year 2010. QUARG considered whether or not such a reduction would be enough to bring average summer PM<sub>10</sub> levels down below the EPAQS (50µg/m<sup>3</sup>) recommendation, and concluded that *"it seems certain that a level of control of secondary particles well in excess of 39% will be needed"* to achieve such a goal. Additional measures that may further reduce secondary particles are in the pipeline, and these include a new UNECE nitrogen protocol, and planned revisions to the EU Large Combustion Plant Directive.

Turning to primary particles, the key factors affecting future particulate emissions from vehicle exhausts are:

- road usage;
- EU regulations designed to reduce particulate emissions - these include: 91/441/EEC (implemented 1992) limiting emissions of new diesel cars; 91/542/EEC (1993) limiting emissions from diesel trucks and buses over 3.5 tonnes; 93/59/EEC (1997) limiting emissions from diesel vans; 94/12/EEC (1997) with new emission limits for petrol and diesel cars;
- the market penetration of diesel cars (assumed to remain at 20% until 2010).

Taking these factors together, models predict that primary emissions of PM10 from road transport will follow the pattern depicted in **Figure 5**, decreasing by ~52% by the year 2010. However, QUARG have estimated that a reduction of at least 60% is required “*if the majority of central urban background sites are not to exceed 50 $\mu\text{g}/\text{m}^3$ ”* in winter. Moreover, much greater reductions in traffic emissions of PM10 may be required from time to time to cope with occasional pollution episodes. For instance, QUARG estimated that reductions of **particulates from vehicle exhausts in the order of 80% would have been required to keep average 24 hour PM10 levels below 50 $\mu\text{g}/\text{m}^3$  during the London pollution episode of 1991.**

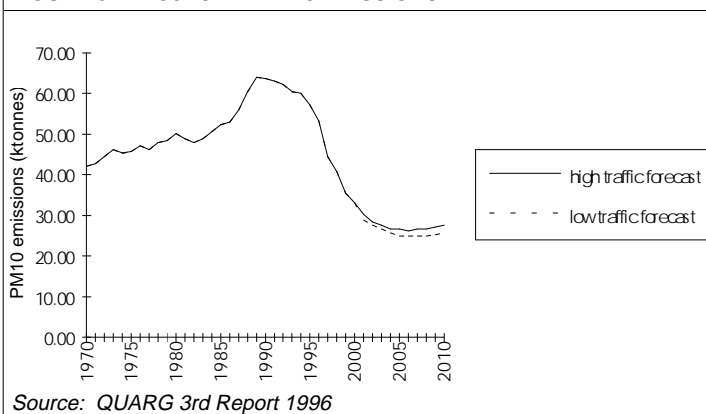
QUARG thus concluded that “*measures currently in place will not deliver a sufficient reduction in primary emissions to ensure that all concentrations fall within the EPAQS recommended limit, even by the year 2010*”. One source highlighted by QUARG was diesel vehicles, with the Review Group pointing out that projections of future PM10 emissions were based on a modest level (20%) of market penetration by diesel cars. Any increase in market share would “*inevitably make matters worse as the current technology diesel car emits far more particulate matter than the modern petrol car*”, and although technological development will narrow the gap, “*there is no current prospect of the diesel improving beyond the petrol car*”.

If current and planned measures appear unable to deliver the improvements desired, what additional measures might be considered? Measures to control emissions of particulates (and other pollutants) from vehicle exhausts were considered by the Royal Commission on Environmental Pollution (RCEP) in its 18th Report. RCEP noted the difficulties in controlling PM10 emissions from diesels - there is a trade-off between decreasing PM10 emissions through improved engine design and increasing NO<sub>x</sub> emissions. Particulate traps are available, but are currently too bulky to be fitted to small vehicles. RCEP thus recommended:

- European vehicle and catalyst manufacturers to pool their research in this area in a programme funded by the European Commission.
- an immediate study to determine which categories of vehicles not designed to the latest standards justify retrofitting with particulate traps, and whether government grants should be offered;
- the Government to press within the EU for more stringent limits for vehicle particulate emission;
- the introduction of fiscal measures to encourage heavy vehicles to meet tighter emission standards.

The European Commission has also examined ways of reducing levels of primary particles (and other pollutants) through its Auto-Oil Programme, set up in con-

**FIGURE 5 PROJECTED PM10 EMISSIONS**



junction with the European motor and oil industries. This project has identified three main approaches - better inspection and maintenance of vehicles; tighter emission standards; and changes in fuel quality - as being the most cost-effective means of reducing vehicle pollution to allow air quality objectives to be met in the next 15 years. A draft directive based on the project findings was agreed by the Commission in June 1996, which in addition to the three approaches, included a proposal to fit all new cars with sensors alerting the driver if the car exceeds pollutant emission limits.

In the UK, Government strategy has been laid out in two recent reports, ‘Air Quality - Meeting the Challenge’ and ‘Transport; The Way Forward’. These identified three main policy strands to improve air quality:

- **national air quality standards and targets** - e.g. establishing new standards and targets for specific pollutants, taking into account both expert advice (from EPAQS) and cost considerations, further improvement of the monitoring network, etc.;
- **local air quality management** - establishing Air Quality Management Areas in local pollution ‘hotspots’ and placing powers and duties on Local Authorities (and other relevant bodies) to meet air quality targets in these areas;
- **vehicle emissions** - e.g. new standards for technology, emissions and fuels, local transport and planning initiatives to reduce the need to travel and encourage use of less polluting types of transport, and tighter enforcement of emissions regulations (targeting the most-polluting vehicles).

However, while this approach has been welcomed, some see the need for more rapid progress. For instance, the National Society for Clean Air (NSCA) has urged the Government to accept the recommendations of both EPAQS (for a PM10 standard) and RCEP (particularly over the need for fiscal measures, see above) without delay. But even then, what powers might be used to prohibit or **restrict traffic through cities on days when the standards are exceeded** remain a source of ambiguity and contention. As pointed out in earlier POST reports, there is little evidence that appeals for

voluntary restraint by drivers have any significant effect, and other countries have found it necessary to restrict vehicle access to towns and cities on high air pollution days (in this context, the French Parliament is debating a bill to make public transport free on days when air quality falls below specified levels). In the UK, local authorities now have the power to use traffic regulation orders as a means of "assessing or managing the quality of air" under the 1995 Environment Act. However, to date no local authority has attempted to use such powers.

## IN SUMMARY

The scientific consensus points to elevated levels of fine particles leading to early deaths and increased respiratory disease. Such effects are detected in UK cities at levels typically found in urban areas, and may be involved in hundreds or some thousands of cases each year across the country. While much of the effect may be to bring forward the day of death for already ill or weak people by only a short time, there appear to be sufficient numbers who die with a life expectancy otherwise measured in years to give measurable differences in mortality rates between cities with different levels of airborne particles. The UK Government has yet to accept a recommendation of an expert group that levels of PM10 should not exceed 50 averaged over 24hrs; meanwhile attention is turning to whether PM10 is the right measure, with evidence suggesting the finer fractions (e.g. PM2.5) are more significant. By far the dominant source of fine particles in urban areas is vehicles (especially diesels), and while improvements can be expected from measures currently in train, additional measures to reduce emissions and/or restrictions on vehicle use during pollution episodes would be needed if recommended standards were to be met. A UK Air Quality Strategy is currently being prepared by the Government and is expected to be released for full consultation in summer 1996.

## FURTHER READING

- AEA Technology, 1995. "Air Pollution in the UK: 1994", NETCEN, AEA Technology, Harwell, Oxon.
- COMEAP, 1995. "Non-Biological Particles and Health", Department of Health, London.
- Dockery, DW and Pope, CA, 1994. "Acute Respiratory Effects of Particulate Air Pollution", Annual Review of Public Health, **15**, 107-132.
- DoE, 1995. "Digest of Environmental Statistics", No 17.
- EPAQS, 1995. "Particles", DoE, London.
- HEI, 1995. "Particulate Air Pollution and Daily Mortality: Replication and Validation of Selected Studies", Health Effects Institute, HEI, Cambridge, MA.
- IP, 1995. "Health Aspects of Particulates", Institute of Petroleum Workshop, June 1995, IP, London.
- LAQN, 1995. "Air Quality in London, 1994", 2nd Re-

- port of the London Air Quality Network, South East Institute of Public Health, Tunbridge Wells, UK.
- NRDC, 1996. "Breath-taking - Premature Mortality Due to Particulate Air Pollution in 239 American Cities", National Resources Defense Council, New York.
- Pope, CA, *et al*, 1995. "Particulate Air Pollution as a Predictor of Mortality in a Prospective Study of US Adults", American Journal of Respiratory Critical Care Medicine, **151**, 669-674.
- POST, 1994. "Breathing in Our Cities - Urban Air Pollution and Respiratory Health"; also Transport: Some Issues in Sustainability (1995).
- QUARG, 1993. "Diesel Vehicle Emissions and Urban Air Quality", 2nd Report of the Quality of Urban Air Review Group, DoE, London.
- QUARG, 1996. "Airborne Particulate Matter in the UK", 3rd Report of the QUARG.
- RCEP, 1994. "Transport and the Environment", 18th Report, HMSO, London.
- Schwartz, J, 1994. "Air Pollution and Daily Mortality: A Review and Meta Analysis", Env. Res., **64**, 36-52.
- Schwartz, J, 1994. "What are People Dying Of on High Air Pollution Days?", Env. Res, **64**, 26-35.
- Dept. of Transport, 1996 "Transport - the Way Forward".
- WHO, 1995. "Update and Revision of the Air Quality Guidelines for Europe", Mtg of the Working Group 'Classical Air Pollutants', WHO, Copenhagen.
- Wordley, J, Walters, S and Ayres, JG, 1996. "Short-Term Variations in Hospital Admissions and Mortality and Particulate Air Pollution", in press.

## GLOSSARY

<b>BS</b>	Black Smoke
<b>COMEAP</b>	C'ttee on Medical Effects of Air Pollution
<b>COPD</b>	Chronic Obstructive Pulmonary Disease
<b>DoE</b>	Department of the Environment
<b>EPAQS</b>	Expert Panel on Air Quality Standards
<b>EUN</b>	Enhanced Urban Network
<b>HEI</b>	Health Effects Institute
<b>H(M)GV</b>	Heavy (Medium) Goods Vehicles
<b>NAEI</b>	National Atmospheric Emissions Inventory
<b>NETCEN</b>	National Environmental Technology Centre
<b>NOx</b>	Oxides of Nitrogen (principally)
<b>PM10</b>	Particulate Matter (diameter < 10µm)
<b>PM2.5</b>	Particulate Matter (diameter < 2.5µm)
<b>QUARG</b>	Quality of Urban Air Review Group
<b>RCEP</b>	Royal Commission on Environmental Pollution
<b>SO<sub>2</sub></b>	Sulphur Dioxide
<b>SPM</b>	Suspended Particulate Matter
<b>TSP</b>	Total Suspended Particles
<b>UFP</b>	Ultra Fine Particles
<b>UNECE</b>	UN Economic Council for Europe
<b>US EPA</b>	US Environmental Protection Agency
<b>WHO</b>	World Health Organisation

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