

Review of the potential health effects associated with particle emissions from gas-fired glass furnaces

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Rates of particle emission from glass furnaces exceed those allowed under the relevant process guidance note. The emitted particles, however, are largely composed of sodium sulphate, which being highly soluble and already present dissolved form in blood and tissue, would not be expected to have a substantial effect on the respiratory health of exposed individuals. This independent review was undertaken to assess the potential health effects that might be associated with particle emissions from gas-fired glass furnaces.

Dispersion modelling suggests that both short and long term increments in local concentrations of airborne particles associated with emissions from glassworks are small in comparison the UK's air quality objectives. Increments in the concentrations of specific toxic components of the waste gas dust including heavy metals and silica are extremely small in comparison to recognised "safe" levels of exposure. Emissions of sulphur dioxide, however, may give rise to a small increase in local levels of aerosol acidity and emissions of nitrogen oxides may give rise to occasional exceedences of the one hour nitrogen dioxide air quality standard.

There is relatively little information available about the potential toxic effects of exposure to airborne sodium sulphate. Experimental results suggest that the toxicity of inhaled sulphate salts varies with their acidity. Fine particles of sodium sulphate are likely to be less biologically active than the more acid sulphates typically present in urban aerosol. The transition metals content of ambient urban PM₁₀ may play an important role in promoting oxidative damage to the airways. The transition metals content of emissions from glass works is generally smaller than that of ambient PM₁₀ and may also be less bioavailable as most of the metals present are locked up in glass. Epidemiological studies have suggested an association between ambient concentrations of airborne sulphate and a range of health endpoints. It seems likely, however, that sulphate in these studies is acting as a marker for some other more toxicologically relevant component of airborne particulate. There is more consistent evidence that aerosol acidity may have an independent effect from that of particles more generally. Overall, it seems likely that primary particle emissions from gas-fired glass furnaces have a lower toxicity per unit mass than typical urban PM₁₀. The formation of secondary acid aerosol under conditions of particularly unfavourable dispersion may occasionally, however, give rise to a small increase in respiratory symptoms in the local area. There would be a greater environmental benefit in cutting emissions of sulphur dioxide and nitrogen oxides than in cutting particle emissions.

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SUMMARY

The glass industry has concerns about the potential health impact of particle emissions from gas-fired furnaces. Rates of particle emission from these furnaces exceed those allowed under the relevant process guidance note. The emitted particles, however, are largely composed of sodium sulphate that would not be expected to have a substantial effect on the respiratory health of exposed individuals. Sodium sulphate is highly soluble and both sodium and sulphate are present in substantial quantities in both body fluids and tissue. Concentrations of more toxic minor components of the waste gas dust including heavy metals and crystalline silica are far too small to be expected to have any association with adverse health effects. This aim of this review was to assess the potential health effects that might be associated with particle emissions from gas-fired glass furnaces with particular reference to the potential of sodium sulphate to cause adverse respiratory effects.

Dispersion modelling suggests that the long term increment in local concentrations of airborne particles associated with emissions from Pilkington's St Helens glassworks is extremely small. Under conditions of extremely poor dispersion the maximum increment in daily mean concentrations associated with emissions from glassworks is only likely to be about $3 \mu\text{g m}^{-3}$. Emissions from glassworks are unlikely to contribute significantly to exceedences of the UK's air quality objective for airborne particles as PM_{10} .

The association between exposure to airborne particles, specifically ambient PM_{10} , and adverse health effects is well established and ultrafine ($<0.1\mu\text{m}$) particles appear to be particularly associated with inflammation. Particles emitted from glass furnaces, however, while respirable, have a much smaller ultrafine component than typical urban traffic pollution. There is relatively little information available about the specific potential toxic effects of exposure to airborne sodium sulphate. Experimental results suggest that the toxicity of inhaled sulphate salts varies with their acidity. Fine particles of sodium sulphate are likely to be less biologically active than the more acid sulphates typically present in urban aerosol. The transition metals content of ambient urban PM_{10} may play an important role in promoting oxidative damage to the airways. The transition metals content of emissions from glass works is generally smaller than that of ambient PM_{10} and may also be less bioavailable as most of the metals present are locked up in glass spherules. Overall, it seems likely that primary particle emissions from gas-fired glass furnaces have a lower toxicity per unit mass than typical urban PM_{10} .

Epidemiological studies have suggested an association between ambient concentrations of airborne sulphate and a range of health endpoints. It seems likely, however, that sulphate in these studies is acting as a marker for some other more toxicologically relevant component of airborne particulate such as $\text{PM}_{2.5}$ or aerosol acidity. Unfortunately there have been no epidemiological studies that have specifically addressed the relative toxicity of the soluble salt versus soot components of urban PM_{10} . It is therefore difficult to estimate the relative risks of adverse health effects associated with exposure to sodium sulphate aerosol as opposed to ambient PM_{10} . Inhaled sodium sulphate particles are likely to grow within the respiratory tract through hygroscopic absorption of moisture and will therefore not penetrate as deeply into the respiratory system as typical particles of ambient PM_{10} . Exposure to sodium sulphate and other soluble salts may therefore be more strongly associated with upper rather than lower respiratory symptoms.

Emissions of sulphur dioxide from gas-fired glass furnaces are substantial in relation to those of primary particles. It seems probable that during the first few seconds to minutes following

emission, a proportion of this SO₂ will condense with water in the waste gas stream to form sulphuric acid and other secondary sulphate species. Ground level concentrations of aerosol acidity are, however, likely to be small in comparison to the levels of acidity associated with adverse effects in both epidemiological and experimental studies. Under conditions of particularly poor dispersion, it is possible that the local levels of acidity might be sufficient to give rise to respiratory symptoms in a few susceptible individuals.

1. INTRODUCTION AND AIMS

1.1. INTRODUCTION

The glass industry has concerns about the potential health impact of particle emissions from gas-fired furnaces. Rates of particle emission from these furnaces exceed those allowed under the current process guidance note (Department of the Environment). The emitted particles, however, are largely composed of sodium sulphate. Sodium sulphate is a highly soluble salt and both sodium and sulphate are present in body fluids at relatively high concentrations in comparison to the likely uptake from air pollution. Sodium sulphate would therefore be expected to have a smaller effect on the respiratory health of exposed individuals than other components of ambient airborne particle pollution. Emissions also include a small amount of crystalline silica and some heavy metals. Both crystalline silica and heavy metals are strongly associated with adverse health effects, but at levels of exposure greatly exceeding those likely to arise from glass furnace emissions.

This review involved an examination of the relevant literature to assess the potential health effects that might be associated with particle emissions from gas-fired glass furnaces with particular reference to the potential of sodium sulphate to cause adverse respiratory effects. The review specifically considered emissions from three stacks in St Helens belonging to glass furnaces operated by Pilkington plc. The review was separated into three strands. Firstly an assessment was made of the impact of particle emissions from gas-fired glass furnaces on local air quality. Secondly, an assessment was made of the potential health hazards associated with the insoluble (heavy metal and silica) component of particle emissions. Thirdly, an assessment was made of the potential risks to health that may be associated with sodium sulphate particle emissions. This third strand constituted the major part of the study.

1.2. BACKGROUND

There is considerable evidence that ambient air quality is associated with a range of health effects and that adverse effects are more consistently associated with concentrations of airborne particles than other pollutants. Episodes of high particle concentrations are associated with increased rates of daily mortality and hospital admission for cardiorespiratory illness, impairments in lung function and increased reporting of respiratory symptoms. The possible long term effects of exposure to airborne particles include a slight reduction in life expectancy, increased risks of bronchitis and respiratory symptoms such as chronic cough and small decrements in lung function. Adverse effects have been associated with very low levels of particle exposure ($<100\mu\text{mg}^{-3}$). The World Health Organization (WHO) have summarised the available dose-response information linking health effects in human to ambient concentrations of PM_{10} (Tables 1.1 and 1.2; information from WHO-Europe website). It can be seen that the effects are generally small and that there is considerable uncertainty associated with the available dose-response information. Causes of uncertainty include interindividual differences in exposures to particles and in susceptibility to the effects of air pollution.

Table 1.1 Dose-response relationships for acute effects associated with exposure to a $10\mu\text{gm}^{-3}$ increase in airborne particles as PM_{10} or $\text{PM}_{2.5}$

Endpoint	Relative risk for $\text{PM}_{2.5}$ (95% confidence interval)	Relative risk for PM_{10} (95% confidence interval)
Bronchodilator use		1.030 (1.020, 1.041)
Cough		1.036 (1.020, 1.052)
Lower respiratory symptoms		1.032 (1.018, 1.046)
PEF* change (relative to mean)		-0.13% (-0.17, -0.09%)
Respiratory hospital admissions		1.008 (1.005, 1.011)
mortality	1.015 (1.011, 1.019)	1.007 (1.006, 1.009)

*Peak expiratory flow

Table 1.2 Dose response relationships for effects associated with long term exposure to a $10\mu\text{mg}^{-3}$ increase in airborne particles as $\text{PM}_{2.5}$ or PM_{10}

Endpoint	Relative risk for $\text{PM}_{2.5}$ (95% confidence interval)	Relative risk for PM_{10} (95% confidence interval)
Mortality (Dockery <i>et al</i> , 1993)	1.14 (1.04, 1.24)	1.10 (1.03, 1.18)
Mortality (Pope <i>et al</i> , 1995)	1.07 (1.04, 1.11)	na
Bronchitis	1.34 (0.74, 1.99)	1.29 (0.96, 1.83)
% change in FEV_1 *children	-1.9% (-3.1%, -0.6%)	-1.2% (-2.7%, -0.1%)
% change in FEV_1 adults		-1.0% (na)

*Forced expiratory volume in 1 second

The mechanisms underlying the association between air pollution and health are relatively poorly understood. High pollution episodes are thought to lead to airways inflammation, giving rise to bronchoconstriction in susceptible individuals such as asthmatics and associated adverse health effects (Seaton, personal communication, 2000). Oxidative damage to airways epithelium can result from exposures to oxidative gases such as ozone and to a lesser extent nitrogen dioxide (Muller *et al*, 1998). Oxidative damage is also associated with particle inhalation which triggers the production of reactive oxygen species in a response that has evolved to defend the lung against inhaled microbes (Driscoll, 1996). Bioavailable transition metals in/on inhaled particles may catalyse the production of superoxide species within the lung to give a similar effect to that of oxidant gases (Donaldson *et al*, 1999, Costa, 1999). Long term exposure to air pollution is thought to lead to chronic inflammation of the airways leading to excessive production of mucus and bronchitis or to degradation of alveoli and emphysema (Seaton, personal communication, 2000). These conditions lead to long term loss of lung function. Similar effects are seen in smokers who, although mostly dying from heart disease, cancer and stroke, also show a more rapid decrease of lung function and much increased risk of death at any age than nonsmokers (Evans and Wolff, 1996). Some of the loss of lung function associated with exposure to air pollution may be reversible, but individuals who are occupationally exposed to particles or who are smokers do not appear to recover all of the lost lung function on cessation of exposure. Small average responses in a large group are likely to be the result of large responses in a small number of susceptible individuals (Lipfert, 1994).

The extent to which components of urban PM_{10} are specifically toxic is not well understood. Animal experiments have shown that exposure to excessive quantities of insoluble inert dust is associated with chronic inflammation leading to the development of fibrosis (Driscoll, 1996). In terms of mass dose, such effects would only be expected at dust concentrations 200-500 times greater than ambient particle concentrations (Yu, 1996). Recent studies have

suggested that particle surface area rather than mass or volume is the main determinant of the onset of inflammation (eg HSE, 1999). Urban particulate contains a substantial ultrafine component and in terms of surface area equivalency, concentrations of urban PM₁₀, would be tens rather than hundreds of times smaller than those associated with the onset of inflammation in animal experiments with inert dusts. Ultrafine particles (<0.1µm) appear to pose special problems for the lung and provoke an acute inflammatory reaction (Donaldson *et al*, 1999). Unlike larger particles, a substantial proportion of ultrafine particles are absorbed by the interstitium rather than being cleared by macrophages. Ultrafine particulate appears to interfere with cell function leading to the release of possibly inappropriate messenger biochemicals (Donaldson *et al*, 1999). Only limited preliminary epidemiological investigations have been made of the specific role of ultrafine particles in the relationship between urban particulate and health. A German study of asthmatic subjects found that lung function parameters were more strongly associated with ultrafine particles than with PM₁₀ as a whole (Peters *et al*, 1997a).

Surface properties are also an important control on the toxic effects of particles in the lung (Richards, 1999). Animal experiments have shown that transition metals sorbed on particle surfaces greatly increase oxidant activity with effects being correlated with the ionisable and soluble metals content of aerosols (Amdur, 1996). Experiments with residual oil fly ash have also highlighted the potential importance of readily leached transition metals (Fe, Ni, V) in causing adverse effects (Dreher *et al*, 1997). Other substances on the surfaces of airborne particles such as PAHs and aldehydes adsorbed on diesel soot may also be pro-inflammatory (Holgate, 1999).

There has been considerable discussion as to the role of particle acidity in causing adverse effects. The calculated effects of acid deposition are far too small for substantial damage to occur at the concentrations of acid aerosol that occur even in highly polluted urban air (American Thoracic Society review, 1999). It is possible, however, that as acid deposition will be concentrated in particular regions, that localised damage to airway mucus and underlying epithelium may occur.

There are many differences between typical urban PM₁₀ such as implicated in the epidemiological studies that show associations between airborne particles and health and the sodium sulphate particles emitted by glassworks. The main issues addressed by this review are whether sodium sulphate particles are likely to be as biologically active as typical urban PM₁₀ and the likely magnitude of any health effects associated with increments in concentration of sodium sulphate particles in comparison with those associated with PM₁₀.

1.3. AIMS

The overall aim of the literature review was to assess the potential for particle emissions from gas-fired glass furnaces to cause adverse health effects. The specific objectives were to:

1. Determine whether overall PM₁₀ emissions from gas-fired glass furnaces add significantly to ground level concentrations of PM₁₀;
2. Determine whether the concentrations of the minor components of particulate emissions (specifically lead, chromium and silica) at ground level are sufficient to give rise to a significant health hazard;
3. Investigate from published sources the bioavailability of heavy metals and crystalline silica incorporated in glassy beads and large grains;

4. Investigate the potential hazards (if any) associated with inhalation of sodium sulphate;
5. If appropriate, suggest a programme of *in vitro* testing to establish the relative toxicity of sodium sulphate in comparison to other particulate substances found in ambient air.

2. METHODS

Relevant published epidemiological and toxicological studies and reviews were identified using standard literature search techniques including the use of appropriate CD ROM databases such as "Pollution and Toxicology" and on-line search facilities. Copies of relevant studies identified by the literature search which were available as journal articles were obtained. Pilkington plc provided a substantial quantity of information about the physical and chemical characteristics of particle emissions from gas-fired furnaces. They also provided information about the modelled concentrations of emitted particles at ground level in the areas impacted by emissions from their St. Helens gas-fired furnaces. These furnaces are believed to be typical of those used generally within the industry.

The assessment of the impact of emissions from glassworks on air quality assumed that the proportion of the insoluble components in the modelled increments in concentrations of airborne particles at ground level was the same as in the primary emissions. It was also assumed that emissions were in the PM₁₀ size range. Background information about air quality around Pilkington's plants in St Helens was obtained from the National Air Quality Archive (<http://www.environment.detr.gov.uk/airq/aqinfo.htm>) and NETCEN (1998). Some consideration was given to the potential hygroscopic growth of sodium sulphate particles following emission and the effects that would have on local air quality. Glass furnaces emit substantial quantities of sulphur dioxide and nitrogen oxides and the potential formation of secondary particles including the condensation of sulphuric acid around sodium sulphate nuclei was considered.

A limited investigation of the potential health effects associated with exposure to the main components of the insoluble particle emissions was made. The typical concentrations of silica and trace metals in ambient air were determined by reference to sources such as the reviews undertaken by the Quality of Urban Air Review Group (1993, 1996) and the National Air Quality Archive. The modelled concentrations of these components in air due to emissions from gas-fired glass furnaces were compared with those normally present in ambient air. Information from the World Health Organisation (WHO) Environmental Health Criteria Documents was used to assess potential hazard and the likely magnitude of any health risk associated with inhalation of the modelled concentrations of these substances.

The potential of sodium sulphate to cause adverse health effects was considered from several viewpoints. The articles reviewed included toxicological and both workplace and environmental epidemiological studies of sodium sulphate specifically. Much of the information reviewed, however, was about the other types of inhaled soluble sulphate particles present in ambient air. Ambient air typically contains low concentrations of ammonium sulphate, sulphuric acid and other sulphate particles that form as secondary particles through the reaction of sulphur dioxide with other pollutants present in urban air. The magnitude of any reported associations between effects associated with the experimental exposure of cells, animals or humans to sodium sulphate and other sulphate salts were reviewed. This allowed an assessment of the toxicity of sodium sulphate in relation to other sulphate compounds. A comparison was also made between the reported health effects associated with sulphate salts and those associated with a selected number of insoluble dusts, most notably urban PM₁₀. An assessment was made as to whether the results of epidemiological studies provide any evidence that exposure to soluble particles in ambient air is associated with a greater or lesser hazard than exposure to less soluble particles.

The information from the various strands of investigation were integrated to make an overall assessment of the likely hazard to health associated with particle emissions from glass

furnaces. An assessment was also made of the potential risks to health associated with the formation of secondary particulate species from glass furnace emissions and limited commentary given on the other potential health effects associated with gaseous emissions from glassworks.

3. CHEMICAL AND PHYSICAL PROPERTIES OF EMISSIONS FROM GAS-FIRED FURNACES

3.1 INTRODUCTION

Pilkington plc have made an extensive investigation of the nature of the particles emitted within their waste gas stream. The main source of particles is from the volatilisation of metals and metal compounds, particularly sodium, from the glass melt. Volatilised sodium, mostly as sodium hydroxide, reacts with sulphur oxides in the flue gases to form droplets of sodium sulphate that crystallise to sodium sulphate dust. Generally there is a substantial excess of sulphur dioxide in flue gases relative to sodium. Small amounts of other compounds may form, particularly following volatilisation of heavy metals from the melt. In addition, the raw materials used in glass production contain fractions with particle sizes less than 20 μm that can be carried over and become entrained in flue gases. These particles are dominated by small particles of crystalline silica derived from the silica sand used in glass making. Generally, less than 5% of flue gas dust comes from carry over. There is also a small population of particles derived from degradation of refractory surface within the furnace structure which include some with a relatively high chromium content. This chapter provides a summary of the particle characterisation information provided by Pilkington. A very brief description of the gaseous components of the waste gas stream is also given as interactions between particles and gases may be important both during transport within the stack and following emission into the atmosphere.

The samples collected by Pilkington have been taken from close to the base of the stack and emitted particles would generally spend approximately a further five seconds travelling up the stack within flue gases before emission. The extent to which particle characteristics are modified during transport within the stack is not known, but some relevant considerations are discussed briefly at the end of this chapter.

3.2 PARTICLE SIZE AND SHAPE

3.2.1 Particle size

In terms of particle number, the modal size range of particles is less than 1 μm . There is some variation in particle size between plants, but the significance of this is unclear. It may reflect slight differences in sample collection methodology rather than necessarily reflecting differences in particle growth history. Differences in furnace design may give rise to some variation in the size and morphology of emitted particles (Table 3.1). A number of the samples show a bimodal size distribution in terms of particle number with a fine mode at about 0.3 μm and a slightly coarser mode at about 1 μm .

Table 3.1 Particle size information (μm) for emissions from glass furnaces (physical size assessed from scanning electron microscopy) as reported by Pilkington in a series of internal reports (Slade, 1997a, 1998a, b, c, 1999a, 2000)

Average	Float glass		Rolled plate		Container glass	
	gas UK	gas Italy	gas	oil	gas	gas
	0.72	0.97	1.07	1.72	1.1-2.5	2.5
standard dev.	0.48	0.04		0.18		
modal range		0.6-0.8	0.4-0.6	0.8-1.0	0.2-0.4	0.2-0.4
range		0.0-4.4	0.0->3.8	0.0-10.4	0.0->6.8	0.0->6.8

If the particle size distribution was recalculated in terms of mass, then both the mean and mode would be slightly coarser. The very small number of particles that exceed $10\mu\text{m}$ in diameter will constitute a disproportionately high percentage of the mass.

The insoluble matter generally has a larger particle size than the soluble and contains particles exceeding 1mm in size. The larger fragments ($>10\mu\text{m}$) are grains of sand and refractory materials and include fragments of quartz (crystalline silica).

3.2.2 Particle shape

Most of the particles have near spherical shapes (Fig. 3.1). The spheres include sodium sulphate and also silicate glass beads. The larger particles including sand grains and fragments of refractory material have much more irregular morphologies (Slade, 1997b, 2000).

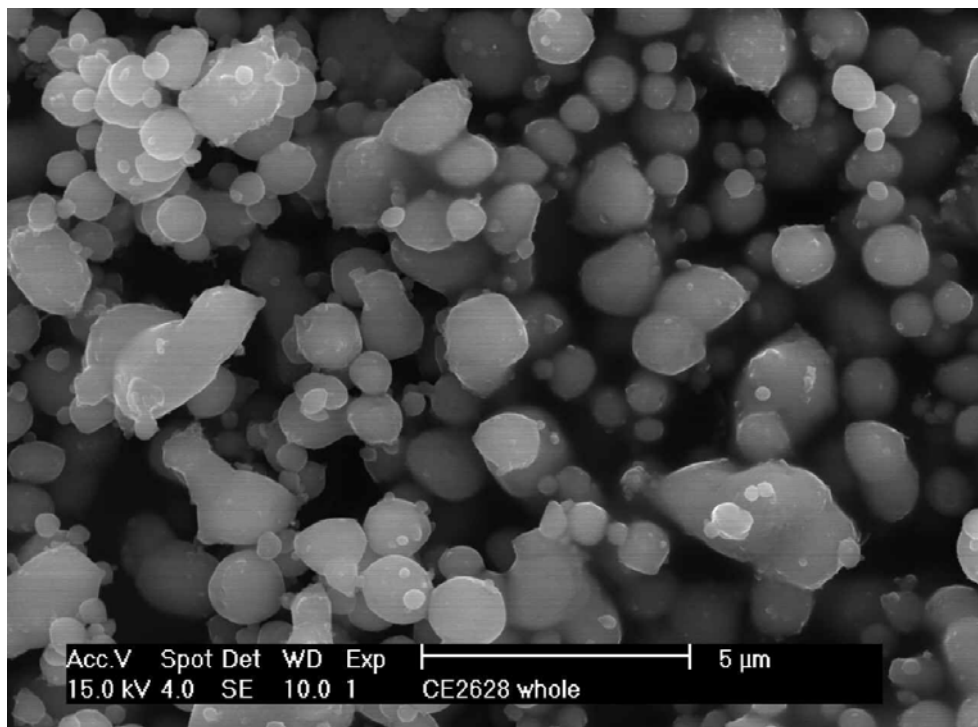


Figure 3.1 Scanning Electron Micrograph of particles emitted from a float glass furnace (supplied by Simon Slade of Pilkington plc)

3.2.3 Comments

Particles in waste gas dust samples are generally within the “accumulation” size range (0.1 μ m-1 μ m) rather than the “nucleation” range (normally <0.1 μ m). Airborne particles “accumulate” by coagulation and by absorbing gaseous material with growth being most rapid and losses due to evaporation being least for particles of 0.1-0.3 μ m (QUARG, 1996).

The spherical shape of particles in the waste gas stream is consistent with their formation as droplets and suggests that particle growth was completed at temperatures greater than the freezing point of sodium sulphate. As temperature falls within the waste gas stream, it is likely that a critical point is reached where sodium sulphate spontaneously nucleates. The nucleation density would have determined the amount of further sodium available to sustain particle growth by the absorption of volatile species. Particle size might therefore be expected to be inversely correlated with nucleation density. The limited range of particle sizes in most samples may reflect the relatively limited time period over which nucleation and growth from volatile species were able to occur. Some particles are likely to have formed through the amalgamation of earlier formed droplets and these particles may be represented by the 1 μ m mode in bimodal size distribution. The extent to which particles could combine in this fashion would depend on the time period over which particles remain in the liquid phase and the concentration of particles in the gas phase (ie the likelihood of particle-particle interactions occurring). Particle size differences between plants could reflect differences in cooling history. The nucleation density is likely to be a function of the availability of suitable nuclei and the degree of supercooling of volatile species. More rapid cooling will give rise to a greater degree of supercooling before nucleation occurs and a greater nucleation density. The temperature of condensation is related to the H:C ratio of the fuel. The smaller particle size found in emissions from gas-fired furnaces is consistent with the lower temperature of sodium sulphate condensation associated with the higher H:C ratio of gas as opposed to oil (Slade, 1995).

3.3 PARTICLE COMPOSITION

3.3.1 Soluble particulate

Over 80% by mass of particle emissions from glass furnaces consists of water soluble salts, predominantly sodium sulphate (Na₂SO₄). The proportion of soluble material present in the examined samples is considerably greater for float glass emissions than for those from rolled plate and container plants (Table 3.2). This is possibly due to the higher quality of materials used in float glass production and the need for a more carefully managed melt to avoid flaws within the glass. The type of fuel used, gas or oil, is likely to be an important influence as the metals content of oil is much higher than that of gas. Although the proportion of soluble material present varies in emissions from different furnaces, its composition is highly consistent with sodium and sulphate in almost molar proportions (2:1). The sodium sulphate is thought to be present largely as μ m sized spheres.

Table 3.2 Percentage of water soluble material in particle emissions from glass furnaces and composition of that soluble material normalised to 100% (based on Slade, 1997a, 1998a,b, 1999a)

	Float glass				Rolled plate		Container glass	
	oil	gas (UK)		gas (Italy)	gas	oil	gas	gas*
%soluble	95.3	98.5	(98.1 -98.7)	96.5	89.6	86.6	92.2	83.9
sodium	29.4	29.0	(28.4 -29.1)	28.0	28.8	20.0	28.1	26.6
potassium	2.4	2.5	(2.3 -2.4)	0.8	2.2	1.5	1.7	1.2
magnesium	1.1	0.0	(0.0 -0.0)	1.1	2.6	9.7	0.2	0.3
calcium	2.2	0.0	(0.0 -0.0)	1.5	0.9	0.9	3.0	4.3
sulphate	63.8	67.7	(69.0 -67.3)	68.5	65.1	67.9	66.7	67.2
chloride	1.1	0.7	(0.2 -0.9)	0.0	0.1	0.0	0.1	0.4
fluoride	<0.1	0.2	(0.1 -0.2)	0.0	0.1	0.0	0.1	0.1

**following oil*

There are five known polymorphs of sodium sulphate of which form V (thenardite) is stable at room temperature and is the phase initially added to the glass melt. Form III is a higher temperature phase that forms above 212°C that converts to Form I on heating to 250°C. Form II forms on cooling of Form I to 236°C and inverts to Form III on cooling to 215°C. Form I occurs as three different forms, referred to by Slade (1996) as the normal Form I and Forms Is and Is2. The sodium sulphate in glass furnace emissions has been identified by Pilkington as being largely composed of the metastable high temperature phases: Form I, Form Is, Form Is2 and Form II (Slade 1996). Analysis indicates that the sodium sulphate lattice contains a substantial proportion of other metal cations (potassium) and anions (chlorine, fluoride) in addition to sodium sulphate. The presence of these other ions may have helped to inhibit the inversion of the sodium sulphate to a low temperature form. Although polymorphism may affect the toxicity of substances such as silica, it seems highly unlikely that there would be a significant difference in the toxicity of different sodium sulphate phases. All forms of sodium sulphate are highly soluble and inhaled particles of any sodium sulphate are likely to be rapidly modified by hydration and dissolution in the respiratory tract.

Some samples of waste gas dust have been found to contain small amounts of other sodium salts such as $\text{Na}_6\text{CO}_3(\text{SO}_4)_2$ - a mixed sodium carbonate sulphate salt (Slade, 1996). The formation of these species is determined by the chemistry of materials used in the melt and also the thermal history of the flue gases. There is no evidence of any potassium salts at quantities sufficient to detect by XRD (1%).

The soluble component of waste gas dust gives rise to a weakly acidic solution with a pH of between 4.5 and 5 (Slade 2000). This suggests that the sodium sulphate particles may contain some absorbed acidic sulphur species, but the cause of the acidity has not been investigated.

Trace amounts of heavy metals such as arsenic and lead have been found in the soluble fraction of waste gas dust. Generally, however, these metals are concentrated in the insoluble fraction. Soluble lead, where found represented only 0.017% of soluble fraction (Slade, 1998c). Although the quantities of trace metals in the soluble component are at or below levels of detection, they may be more bioavailable than metals locked up in insoluble particles.

3.3.2 Insoluble component

The insoluble component of emissions is reported to contain glassy spherical particles that appear to be of very variable composition (Slade, 1997a). Silicon was generally the most abundant element present (apart from oxygen which was not measured). The silicon was mostly present within the glass spheres but was also present in sand and cullet (recyclable waste glass) fragments carried over from the raw materials and in fragments eroded from the furnace lining.

Most of the glass spheres investigated have silicate compositions that incorporate a range of other components such as potassium, calcium, sodium, titanium, iron, chromium and magnesium in variable proportions. In addition metallic flakes and other metal based particles can be present. Table 3.3 shows an average composition based on four dust samples. Some of the fragments of silica-containing material show evidence of some erosion and alteration within the waste gas stream.

Table 3.3 Elemental composition of insoluble fraction of waste gas dust (Slade, 1997b). Some of the missing mass will be oxygen that was not readily detected using the standard analytical methods available at the time.

Element	Weight percent	Element oxide	Weight percent
silicon	20	SiO ₂	42.9
calcium	7	CaO	9.8
chromium	5	Cr ₂ O ₃	7.3
magnesium	4	MgO	7.2
phosphorous	4	P ₂ O ₅	9.2
sulphur	3	SO ₃	7.5
lead	3	Pb ₂ O ₅	3.2
sodium	3	Na ₂ O	4.0
potassium	3	K ₂ O	3.6
aluminium	2	Al ₂ O ₃	3.8
chlorine	1	Cl	1.0
tin	1	SnO	1.1
vanadium	0.5	V ₂ O ₅	0.9
nickel	0.2	NiO	0.3
titanium	0.1	TiO ₂	0.2

Most of the insoluble component of waste gas dust is vitreous rather than crystalline. The major crystalline phase is alpha-quartz. The quartz content has been found to range from 4.8% to 15.4% and is probably largely derived from the silica sand used as a raw material (Slade, 1997a). Other compounds include phlogopite, calcium magnesium sulphate, calcium phosphate, gypsum, anhydrite, alternate calcium sulphate, bassanite, corundum, haematite, mullite, cassiterite, periclase, calcite and fluorite. All of these minerals are potentially present in trace (<ngm⁻³) quantities in ambient air.

Carbonaceous soot forms a very small component of the insoluble fraction and is confined to some samples from oil-fired furnaces. Some samples of waste gas dust from oil fired furnaces are grey tinged, partly due to the presence of soot. Soot may also provide nuclei for NaSO₄ particles. Generally, however, samples of waste gas dust are pale green and contain little or no soot.

3.3.3 Heavy metals

Heavy metals are present in small quantities within the particulate fraction of float glass emissions. They are generally concentrated within the insoluble component and are thought to be largely contained with the glass spheres. A range of heavy metals have been reported: vanadium, tin, titanium, nickel, lead, chromium, copper and arsenic (Slade, 1997a, b, 1998 a, b, c, 1999). Metals in the fuel may act as sources for particle formation or catalyse condensation reactions leading to the formation of particles. This is particularly true for V, a common contaminant in oil. In general the level of heavy metals is greater for oil fired than for gas-fired furnaces reflecting the greater metals content of the fuel. Higher levels of heavy metals were found in emissions from the rolled plate plant because of the use of cullet containing lead within the input materials to the glass melt. This practice has been stopped at this plant.

Emissions of heavy metals from glass plants are small in comparison to other UK sources (Table 3.4; data from National Emissions Inventory).

Table 3.4 Contribution of the glass industry (all sectors) in 1998 to UK emissions of heavy metals as percentage of total emissions for each metal. Also shown are the contributions from other sources of metal pollution.

	Glass	Road transport	Public Power	Waste incineration	Petroleum refining	Combustion in industry
Cr	11.3	0.0	27.4	0.0	1.6	8.1
Pb	1.3	57.3	2.5	0.3	0.2	21.5
As	2.0	0.0	11.8	0.0	0.0	45.1
V	3.5	0.1	2.7	0.0	48.5	29.4

3.4 FACTORS AFFECTING THE FORMATION OF SODIUM SULPHATE

Pilkington have undertaken extensive investigations of the formation of sodium sulphate in the waste gas stream (Slade, 1995). An important general factor that determines the mass of sodium sulphate formed in the waste gas stream is the availability of sodium. Sulphur is normally in excess over sodium in the waste gas stream, although some may be present as sodium chloride (NaCl). Sodium chloride is a relatively volatile species and contamination of the melt by NaCl can lead to higher sodium concentrations in the waste gas stream than in its absence. In recent years Pilkington have tightened the specification for the sodium carbonate added to the raw materials to reduce NaCl contamination and therefore reduce sodium volatilisation from the melt.

Slade (1995) found that emissions of sodium sulphate are generally 30% lower from gas-fired furnaces than from oil fired despite the melt being at a similar temperature and being of essentially the same composition. This may partly be due to the effect of the H:C ratio of the fuel on the temperature of condensation of Na₂SO₄. Condensate in gas-fired systems forms over a shorter time period than in oil fired systems so that potentially less particulate matter is formed. The differences between oil fired and gas fired systems are most marked at the temperatures where the condensate starts to form. The presence of NaCl may complicate the reaction process, particularly for gas-fired systems where only some of the NaCl will react to form NaClSO₃ before condensing. Sodium chloride may combine with condensing Na₂SO₄ to form a sticky eutectic melt or it may react with gaseous SO_x to form Na₂SO₄. The lower sulphur levels associated with gas firing than with oil firing may mean that less NaCl and less NaClSO₃ is converted to Na₂SO₄. Slade (1995) concluded that the formation of a potentially more sticky Na₂SO₄ condensate under gas firing than oil firing could be an important factor

that led to lower particle emissions. This does suggest a potential method of mitigating particle emissions in that if a component were added to the melt to further increase the stickiness of condensate in the waste gas stream, more would accumulate within the regenerator rather than being emitted. This would, however, lead to the need for more frequent maintenance and would be unlikely to be the best practicable environmental option. In the long term it might be possible to redesign the flue system of new furnaces to take advantage of the potential for trapping particulate by creating a more sticky condensate.

3.5 GASEOUS EMISSIONS

The mass of particle emissions from gas-fired furnaces is relatively small compared with those of sulphur dioxide and nitrogen dioxide (Table 3.5) but emissions of both these pollutants are within the limits set out in the current process guidance note (Department of the Environment, 1995). Both sulphur dioxide and nitrogen dioxide could contribute to the formation of secondary particles and are associated with adverse health effects under some circumstances. Emissions also include the acidic species hydrogen chloride and hydrogen fluoride. These will add to local concentrations of acid aerosol but are not generally thought to be of great importance in the relationship between ambient air quality and health at the levels found in UK air.

Table 3.5 Typical emissions from gas-fired float glass furnaces in St Helens mg/Nm³ at 8% O₂ (wet volumes) – information provided by Pilkington. Actual emissions vary with conditions

	UK5	UK6
SO ₂	721	718
particles	145	163
HCl	26.0	28.2
HF	4.43	4.78
NO _x	2285	2644

3.6 MODIFICATION OF PARTICLE CHARACTERISTICS DURING TRANSIT IN THE WASTE GAS STREAM

The samples of particles from the waste gas stream were collected from the base of the stack and may not be representative of emissions from the top of the stack. Particle emissions are, however, well within the size range most strongly associated with light scattering (0.2-2µm) suggesting that even if some modification of particle size occurs within the stack, emissions are still well within the PM₁₀ size range.

It is highly unlikely that further sodium sulphate condensation occurs at higher levels in the stack as all the sodium present in the waste stream is already locked up in sodium sulphate or other sodium salts. It is possible that some oxidation of sulphur dioxide may occur on particle surfaces to form sulphuric acid or NaHSO₄ with some enlargement of particle size. It seems likely that there would be sufficient water present to sustain sulphuric acid formation, although the rates of acid formation may be such that little would form during the few seconds of transport through the stack. Given the temperature at which waste gases pass through the stack (>350°C), it seems unlikely that the sodium sulphate particles will absorb much water before emission and are likely to be emitted as dry particles rather than as aqueous droplets. The moisture present within emissions may, however, condense around sodium sulphate particles immediately following emission to form droplets and this is discussed further in section 4.7.

In addition to simple particle growth, coagulation is also possible although it is not an important process for salt species present at much lower concentrations in the general atmosphere. The bimodal size of particles in flue gas from gas-fired furnaces suggests that particle coagulation is an important process while particles are in the liquid phase. It may be less important at the lower concentrations that may prevail higher in the stack and when particles are in the solid rather than liquid phase. The amount of moisture present may also affect the potential for particle coagulation as droplets will coagulate more readily than dry particles.

Overall, it seems likely that some modification to the particle size distribution occurs during transit up the stack, but particles are likely to generally remain about one micron in size. Some modification to the size of emitted particles may occur subsequent to emission and this is discussed further below.

4. AMBIENT PARTICLE CONCENTRATIONS ASSOCIATED WITH GAS-FIRED GLASS FURNACES

4.1 INTRODUCTION

This chapter reviews the predicted increments in local levels of air pollutants as a result of emissions from St. Helens Pilkington glassworks. This includes consideration of the total increment in airborne particles assuming no modification of particle characteristics following emission and increments in concentrations of heavy metals and crystalline silica. It also includes consideration of the potential growth of sodium sulphate particles in the atmosphere and the potential formation of secondary particles associated with sulphur dioxide emissions. The first part of this chapter describes some relevant measures of particle concentration and also the typical background concentrations of particles in Lancashire. A brief consideration of the effects of the gaseous emissions on local air quality is included towards the end of the chapter.

4.2 MEASURES OF PARTICLE CONCENTRATIONS

There are a number of different measures of particle concentration and the relationship between these measures depends on local factors. Particle concentrations are conventionally measured in terms of mass, although particle number may be a more toxicologically relevant measure of dose (QUARG, 1996). Recent toxicological work undertaken at the IOM (HSE, 1999) and elsewhere (Oberdorster, 1996; Driscoll, 1996) has suggested that particle surface area may also be a toxicologically relevant measure of dose.

Traditionally in the UK, particulate concentrations were measured in terms of Black Smoke. The measurement was essentially a colorimetric assessment of the soot staining of a filter. Black smoke was also measured in Europe, but using slightly different methods and calibration so that measurements made in the UK are not directly equivalent with those made elsewhere in Europe. During the last decade, the UK has moved increasingly to monitoring of particulate as PM_{10} using the Tapered Oscillating Element Microbalance (TEOM). PM_{10} is approximately equivalent to the ISO thoracic fraction, ie the fraction of airborne particulate that can penetrate to the lung. It represents a log normal sampling efficiency with a median cut off of $10\mu m$ aerodynamic diameter. The TEOM, however, underestimates the mass of PM_{10} present in air relative to measurements made using direct gravimetry (DETR, 1999). One cause of this underestimation is that air taken into the TEOM is heated and it is likely that volatile organic compounds and nitrates are volatilised at this stage and lost to subsequent measurement (QUARG, 1996).

Many of the important epidemiological investigations of the effects of ambient particles on health have been performed in the US where particle concentrations were traditionally monitored in terms of total suspended particles (TSP). Sulphate concentrations were also monitored and provide a useful index of fine particle pollution. More recently the US has adopted an air quality standard for PM_{10} and there is increasing interest in $PM_{2.5}$, which is sometimes referred to as "high risk respirable" (QUARG, 1996). It is thought that the $PM_{2.5}$ sampling convention corresponds closely to the deposition efficiency in lungs of individuals with compromised respiratory health (QUARG, 1996). Workplace exposure to airborne dust is usually assessed in terms of the respirable fraction, approximately PM_4 , which corresponds to the fraction likely to penetration to the deep lung (alveolar space).

4.3 MEASUREMENT OF SOLUBLE PARTICLE CONCENTRATIONS

Specific measurements of different phases in air such as metals or soluble salts involve the analysis of material deposited on filter and are subject to a number of potential artefacts (Lippmann and Thurston, 1996). Levels of acidity may be underestimated where samples are collected on alkaline glass fibre filters as opposed to teflon or quartz filters. Levels of sulphate and nitrate may be overestimated due to the reaction of sulphur dioxide and NO_x on the filter surface or on the deposited samples (Lippmann and Thurston, 1996). These difficulties of measurement may have weakened the power of some studies to find associations between health and soluble particle concentrations. They may also contribute to differences in the findings of studies undertaken in different parts of the world.

4.4 BACKGROUND PARTICLE CONCENTRATIONS IN LANCASHIRE

4.4.1 PM₁₀

The most recent year for which summary information about particle concentrations in the UK is publicly available is 1996. Modelled background concentrations of PM₁₀ in rural areas of Lancashire and Cheshire in 1996 were within the range 17.6-20 µgm⁻³ with concentrations in urban areas being in the range of 20.1-22.5 µgm⁻³ (National Air Quality Archive). Maximum daily concentrations in the nearby urban areas of Liverpool and Manchester exceeded 90 µgm⁻³ and the EPAQS 50 µgm⁻³ standard was exceeded on about 40 days in the year (Table 4.1). Maximum levels of particle pollution and the number of exceedences of the EPAQS standard would be expected to be slightly lower in adjacent rural areas.

Table 4.1 Summary of PM₁₀ concentrations in Liverpool and Manchester (1996)

	Annual mean	98th percentile (hourly average)	99th percentile (daily average)	Maximum hourly average	Maximum daily average	Days > 50µgm⁻³ per year
Liverpool	25	75	74	168	94	37
Manchester	26	76	73	174	96	42

4.4.2 Soluble particle concentrations

The dominant source of sodium salts in UK air is from marine aerosol whereas the dominant source of sulphate is the oxidation of sulphur dioxide pollution. Soluble particle concentrations are not routinely monitored in the UK (NETCEN, 1998) and estimated secondary soluble particle concentrations in the St Helens area are 9 µgm⁻³ (National Air Quality Archive). Measurements made in the 1980s in Lancaster suggest that typical long term mean concentrations of sulphate were between 5 and 9µgm⁻³ (Table 4.2; QUARG 1996). Secondary sulphate formation is most rapid during summertime photochemical events and measurements of sulphate concentrations in Essex show diurnal and seasonal variations consistent with the formation of sulphate through photochemical activity (Tables 4.3, 4.4). Secondary sulphate can also form through the oxidation of sulphur dioxide on particle surfaces which is of particular importance during the winter months when sulphur dioxide concentrations tend to be highest. Measurements made in the early 1990s in Oxfordshire suggest that sulphate and soluble salt concentrations in general were greatest in the winter (7µgm⁻³ compared with 5 µgm⁻³ in summer) when SO₂ concentrations were also highest (Lee *et al*, 1999). Concentrations were lowest in the autumn (2.4 µgm⁻³) when levels of photochemical activity and concentrations of SO₂ were both low.

Table 4.2 Airborne concentrations of ions in soluble particles measured at Hazelrigg – rural Lancaster 1979-81 (24 hour means)

$\text{SO}_4 \mu\text{gm}^{-3}$		$\text{NH}_4^+ \mu\text{gm}^{-3}$		$\text{NO}_3 \mu\text{gm}^{-3}$	
mean	range	mean	range	mean	range
8.78	1.6-52.2	4.32	0.2-26.2	5.45	0.6-20.2

Table 4.3 Airborne concentrations of soluble salts measured in Essex

	$\text{SO}_4 \mu\text{gm}^{-3}$		$\text{H}^+ \text{nmolm}^{-3}$		$\text{NH}_4^+ \mu\text{gm}^{-3}$	
	mean	std*	mean	std**	mean	std
Walton Pier 1987	5.7	4.6	29		2.7	2.4
Essex University 1987	5.4	4.4	18		3.0	2.4
Great Domsey 1987	5.8	4.8	12		3.3	2.8
Essex University 1987	8.3	8.1	17		5.3	5.4
Ardleigh Day Summer 1989	8.5	7.6	18		3.9	3.4
Ardleigh Night Summer 1989	6.9	6.0	14		4.5	3.4

*standard deviation

** not given

Table 4.4 Mean of four sites in Essex (Essex University, Colchester, Dedham, Walton Pier)

	$\text{SO}_4 \mu\text{gm}^{-3}$	$\text{NH}_4^+ \mu\text{gm}^{-3}$	$\text{NO}_3 \mu\text{gm}^{-3}$	$\text{Cl} \mu\text{gm}^{-3}$
autumn 1986	3.47	2.66	3.07	1.09
spring 1987	9.45	5.63	9.04	2.51

Levels of aerosol acidity in the UK are generally less than 100nmolm^{-3} (equivalent to $5 \mu\text{gm}^{-3}$ H_2SO_4 ; QUARG, 1993). Regional variations may reflect differences in the primary source of sulphur pollution and the availability of ammonia (NH_3) for the neutralisation of acid species formed through photochemical reactions. Concentrations of aerosol acidity during the 1950s and 1960s in urban regions of the UK were considerably higher than during more recent decades. In December 1962, for example, the highest daily mean concentration of H^+ , as H_2SO_4 , in London was $347 \mu\text{gm}^{-3}$ (7080nmolm^{-3} H^+), whereas in 1990 the highest daily mean sulphate concentrations (not specifically H_2SO_4) were just under $20 \mu\text{gm}^{-3}$ (QUARG 1993).

Concentrations of both acidity and sulphate tend to be higher during summertime high pollution events in North America than during comparable events in the UK or Europe. Such events also occur more frequently because of the hotter, and sunnier nature of North American summers. The air in which the sulphate forms during photochemical events in North America is well mixed and there is little opportunity for ammonia to accumulate and react with secondary particles of sulphuric acid, giving rise to a more acidic aerosol than typically arises in Europe (Brauer *et al*, 1995). Indoor levels of aerosol acidity are normally very low because of neutralisation by ammonia which suggests an association between health and outdoor levels of aerosol acidity is unlikely for most of the population (Brauer *et al*, 1995).

4.4.3 Heavy metal concentrations

Typical concentrations of heavy metals in UK are extremely low with the highest concentrations of most metals arising in the vicinity of metal works (Table 4.5). Current lead concentrations are probably now lower than shown in Table 4.5 as a result of the phasing out of leaded petrol. Industrial emissions for heavy metals have reduced substantially over the last 30 years and this is reflected in the substantial reduction in airborne concentrations of heavy metals in UK urban areas (National Air Quality Archive). Information held on the National Emissions Inventory suggests that the glass industry as a whole emits a relatively small proportion of the total heavy metals emissions to air in the UK (Table 3.4).

Table 4.5 Typical concentrations of a range of heavy metals in UK air 1986-89 as reported by QUARG (1996) and the National Air Quality Archive

Metal	Ambient Concentrations 1986-1989 ngm ⁻³		Urban concentrations 1998
	urban air	rural air	
Chromium	5.6	0.95	2-5.2
Iron	622	152	380-1255
Copper			10-17.7
Lead	183	33.4	18-54
Arsenic	2.65	1.32	
Vanadium			1.8-4.8

4.4.4 Crystalline silica and other mineral dust

Mineral particles are generally concentrated in the coarse fraction of airborne particulate and constitute only about 20% of the mass of PM₁₀ (QUARG, 1996). Quartz (crystalline silica) is one of the most abundant minerals in the earth's crust and is commonly present in airborne dust. Illite and other clay minerals such as chlorite and kaolinite are also present in airborne dust together with feldspar, calcite, dolomite, gypsum and a range of other salts. Maximum concentrations of quartz are likely to be less than about 2µgm⁻³ in most areas.

4.5 MODELLED INCREMENTS IN PARTICLE CONCENTRATIONS

Pilkington have undertaken dispersion modelling to determine the increment in local concentrations of air pollution that could be attributed to emissions from their St. Helens furnaces (Wright 2000). In earlier investigations they also considered the effects of other major industrial sources of local air pollution: the United Glass Furnaces in St Helens (now closed), Ravenhead Glass, Owens Corning Fibre Glass and Fiddlers Ferry Power Station. The dispersion modelling was undertaken using the US Environmental Protection Agency program ISCST3. Particle size data was not included as an input variable, so the modelled increments in particles do not allow for the effects of particle deposition. This may lead to a slight overestimate of concentrations, particularly if rates of particle deposition are enhanced by particle growth as a result of hydration or formation of secondary sulphate (see below). Given that most of the particles emitted are likely to be within the PM₁₀ size range, for the purposes of this assessment, the suspended particulate matter has been treated as PM₁₀.

The results of the modelling suggest that during the most unfavourable conditions for dispersion, the maximum hourly increment in local concentrations of particles (within a 12 month period) due to the combined effects of the three St Helens furnaces would be 32µgm⁻³ (Table 4.6). This increment would arise in the area of Billinge Hill, about 5km north of St

Helens. In most of the local area, however, the maximum increment would be about $10\mu\text{gm}^{-3}$ (Fig. 4.1). The maximum hourly increment due to any single furnace would be $15\mu\text{gm}^{-3}$. The results of the earlier modelling suggest that during the most unfavourable hour of the year, the maximum increment in local concentrations of particles due to the aggregated sources was $56\mu\text{gm}^{-3}$. The contribution of the four Pilkington group stacks to this peak was $50\mu\text{gm}^{-3}$ but only $24\mu\text{gm}^{-3}$ came from the two float glass sites. Under these conditions concentrations of particles would be raised by more than $20\mu\text{gm}^{-3}$ over an area of about 20km^2 , 5-10km north of St Helens.

Table 4.6 Modelled maximum increments (μgm^{-3}) in airborne particle concentration arising during a 12 month period as a result of Pilkington's stacks in St Helens.

Stack	Highest 1 hour	Highest 24 hours	99 th percentile 24 hour mean	90 th percentile 24 hour mean	Annual mean
CH2	9.3	1.5	1.0	0.5	0.1
UK5	15	1.6	0.9	0.4	0.1
UK6	8.8	1.1	0.5	0.3	0.1
all three	32	3.8	2.0	1.1	0.3

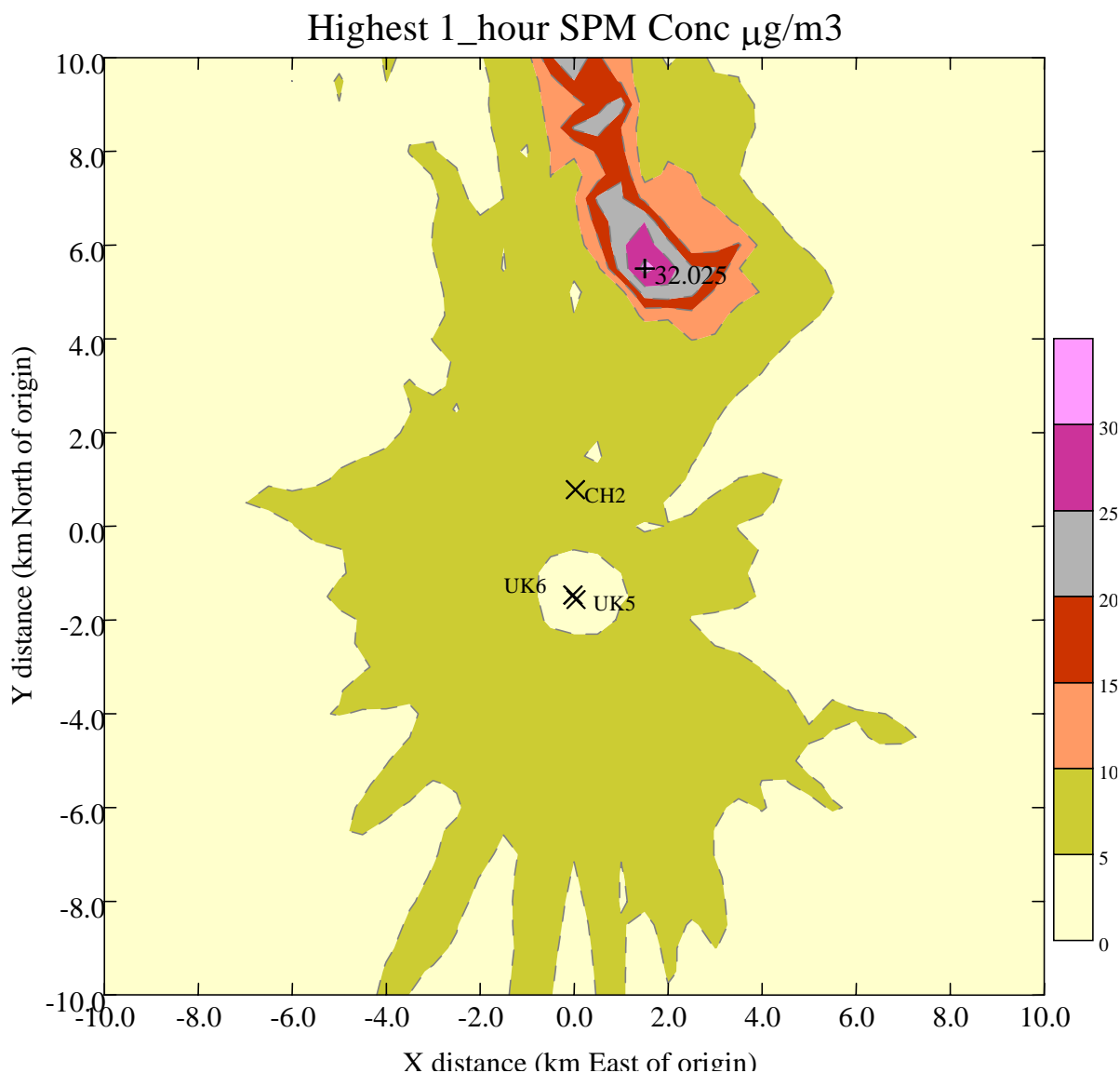


Figure 4.1 Modelled maximum hourly increment in concentrations of airborne particles arising from Pilkington's stacks in St. Helens

Epidemiological research into the health effects associated with particles has generally considered particle concentrations in terms of 24 hour running means, daily averages or longer averaging times. Dispersion modelling suggests that maximum increment in the 24 hour mean particle concentrations due to the three Pilkington float glass furnaces during a typical year would be only $4\mu\text{g}\text{m}^{-3}$, also in the area of Billinge Hill. Under some conditions of poor dispersion, the maximum increment in concentrations ($1\text{-}2\mu\text{g}\text{m}^{-3}$) might arise on the high land to the west of St. Helens rather than to the north. It does not seem likely that the emissions will contribute significantly to exceedences of the EPAQS $50\mu\text{g}\text{m}^{-3}$ standard (24 hour running mean) for ambient PM_{10} . The current UK air quality objective for PM_{10} is that this standard should be exceeded on no more than 35 days in a year (formerly 4 days). The modelled 99th and 90th percentile 24 hour mean increments in particles associated with current emissions from the St Helens glassworks (maximum of $2.0\mu\text{g}\text{m}^{-3}$ and $1.1\mu\text{g}\text{m}^{-3}$ respectively) indicate that these emissions are relatively unimportant in terms of meeting this objective.

In terms of annual mean concentrations of particles, the modelled increments due to the glass furnaces and other industrial sources of PM₁₀ in the St. Helens area are extremely small. The maximum increment in annual mean concentrations in the most impacted area (Billinge Hill) is 0.3µgm⁻³.

Overall, the results of the modelling indicate that the area that should be most affected by particle emissions from Pilkington's furnaces and other local sources of PM₁₀ was Billinge Hill. Pilkington therefore attempted to verify this by undertaking sampling for respirable dust on Billinge Hill (Slade, 1998d). The daily average concentrations of respirable dust measured varied between 25 and 73µgm⁻³, somewhat higher than might have been anticipated from the National Air Quality Archive. The highest concentration arose when the wind was from the North West and therefore could not be attributed to emissions from St Helens. The main source of dust on this day was thought to be agriculture. Concentrations on days when the wind was from southerly directions were 25-50µgm⁻³. The Na:S ratio of dust samples was variable, but showed no consistent relationship with wind direction. Overall there was no clear evidence that emissions from St. Helens had a clear impact on air quality at Billinge Hill, but the survey was of very limited duration. A direct reading instrument such as the TSI Dust Trak could be used to take hourly measurements over a much longer time period which could be analysed in conjunction with local meteorological data to look for an influence from St. Helens. It does seem probable, however, that the effect of emissions from the glass furnaces on local air quality would be swamped by the emissions from road transport within the region with intermittent peaks of concentration at the measurement site being associated with agricultural activities in adjacent fields.

Dispersion modelling of the effects of emissions from other glass furnaces have similarly concluded that emissions have a relatively small effect on local air quality. A Dutch study of emissions from a container glass plant in the Netherlands concluded that the contribution to total deposition of sodium and sulphate in the local area was less than 10% under the worst conditions and generally less than 0.01% (Beerkens *et al*, 1998).

4.6 MODELLED INCREMENTS IN SPECIFIC COMPONENTS OF PARTICULATE EMISSIONS

4.6.1 Heavy metals

The adverse effects associated with environmental exposure to heavy metals are related to long term exposure rather than short term peaks of concentration. Given that the maximum increment in annual average concentrations of PM₁₀ from gas fired glass furnaces is likely to be less than 1µgm⁻³, the increments in concentrations of heavy metals is likely to be extremely small (Table 4.7). Heavy metals emissions from oil-fired plants and from the rolled plate plant are somewhat greater than for gas fired float glass furnace, but are still unlikely to have a measurable impact on local air quality. Overall heavy metal emissions from gas-fired glass furnaces are likely to make a negligible contribution to ambient concentrations of airborne metals (Tables 4.5, 4.7). A Dutch study of the impact of lead emissions from a glass plant in the Netherlands on local air quality similarly showed that lead emissions accounted for a small proportion of local lead deposition (Beerkens *et al*, 1988).

Table 4.7 Estimated increments of airborne heavy metals (ngm^{-3}) for a $1\mu\text{gm}^{-3}$ (1000ngm^{-3}) increment in annual concentrations of PM_{10} associated with a gas-fired float glass furnace (based on analysis of waste gas dust reported by Slade 1997b, 1998c, 1999) compared with typical concentrations in UK air 1986-89 as reported by QUARG (1996) and the National Air Quality Archive

Metal	Gas fired float glass	Oil-fired float glass	Rolled plate	Background concentrations
Chromium	4.0	2.4	0.6	<5
Iron	1.5	2.2	3.3	<1200
Copper	0.4			<18
Lead	0.6	1.2	9.0	<50
Arsenic*	0.2-1.5			<3
Vanadium*	<0.2	0.4	5.0	<5

* quantification of the proportion of arsenic in emissions was less reliable than for other metals

**only present in emissions from gas-fired furnaces that that have previously used oil

4.6.2 Crystalline silica and other minerals

The adverse effects associated with exposure to crystalline silica in the workplace are associated with long term exposure to elevated concentrations of crystalline silica ($>>100\mu\text{gm}^{-3}$). Given that the greatest increment in annual concentrations PM_{10} associated with emissions from glass furnaces is likely to be less than $1\mu\text{gm}^{-3}$, the increment in crystalline silica is likely to be less than 1ngm^{-3} . The concentrations of other mineral phases emitted are likely to be even smaller.

4.7 PROBABLE FATE OF SODIUM SULPHATE PARTICLES FOLLOWING EMISSION

Adsorbed moisture plays an important role in modifying sulphate particle size distribution in clouds, particularly for particles in the submicron size range (Russell and Seinfeld, 1998). Sodium sulphate is a highly soluble salt and particles of sodium sulphate might be expected to rapidly absorb water to become droplets on emission to air. The presence of considerable quantities of moisture in emissions means that the relative humidity within the plume immediately after emission would be somewhat higher than in the general atmosphere. Hygroscopic salt particles do not grow steadily under conditions of increasing humidity, but instead at a critical level of humidity will suddenly absorb water and greatly increase in size – the deliquescence relative humidity (DRH). The DRH for sodium sulphate is higher than of other soluble salts likely to be present in the atmosphere and under most conditions of relative humidity sodium sulphate will exist as dry particles (Ansari and Pandis, 1999). The incorporation of other ions such as ammonium, chloride or nitrate into the sodium sulphate lattice will however greatly reduce its DRH with values as low as 50% being possible (Potukuchi and Wexler, 1995). Interactions between sodium sulphate particles and other components of waste gas emissions and ambient air pollution could therefore influence their DRH with a subsequent effect on particle size.

It seems probable that during periods of relatively high humidity, the mass increment in local aerosol concentrations will be greater than that predicted by the dispersion modelling due to the hygroscopic growth of the emitted particles. Much of the effect may, however, be offset by increased rates of particle deposition (Koutrakis *et al*, 1989; Lazaridis and Koutrakis, 1997; Quinn and Ondov, 1998). At very high humidities a substantial proportion of particles are likely to be washed out in rain and would not contribute to local concentrations of PM_{10} .

Over half the acid sulphur deposition on the UK is in rainfall rather than dry deposition (DETR website). The biological significance of the potential increase in mass of particles is unclear as there is increasing evidence that particle number and/or particle surface area are of more biological relevance than particle mass concentration. There is also growing evidence to suggest that the risks to health associated with airborne particles diminish with increasing particle size.

In addition to particle growth occurring through hydration, the sodium sulphate emissions may also provide nuclei for the condensation of sulphuric acid from sulphur dioxide emissions and this is discussed further below. The formation of aqueous droplets may be of significance in this context in leading to enhanced rates of formation of secondary sulphuric acid (see below).

4.8 POTENTIAL FORMATION OF SECONDARY SOLUBLE PARTICLES INCLUDING ACID AEROSOL

4.8.1 Potential for sulphuric acid formation from emissions

There is a substantial quantity of SO_x in emissions from glassworks that could potentially be oxidised to sulphuric acid either through gas-phase reactions or reactions on or within particles including droplets. About 5% of this SO_x is as SO₃ and it seems probable that most of this SO₃ will be oxidised to sulphuric acid during the first few minutes following emission. Sulphuric acid has a low vapour pressure and condenses readily, even in the absence of nuclei. Particle surfaces including sea salt and carbonaceous soot are particularly important in the winter in catalysing the formation of sulphuric acid whereas during the summer photochemical gas phase reactions dominate (Meng and Seinfeld, 1994, Kerminen *et al*, 1997). Both rates of particle growth and of sulphuric acid nucleation increase as humidity increases, but the relative acidity of particles drops sharply (Gupta *et al*, 1995). Any sulphuric acid formed is likely to be rapidly converted to ammonium sulphate (Lee *et al*, 1999).

The emission of sodium sulphate particles from glassworks may enhance sulphuric acid condensation by providing a source of nuclei and particle surfaces and much of the secondary sulphate formed is likely to be present in mixed sodium-ammonium-sulphuric acid salts. In addition trace amounts of soot and metals in the emissions may enhance the condensation of sulphuric acid as may the presence of particulate pollution from other sources. The quantities of sulphuric acid formed directly as a result of photochemical oxidation (Lazaridis and Koutrakis, 1997) or on days of high humidity through the diffusion of SO₂ into water droplets (Kerminen *et al*, 1997) are likely to be much smaller. The rate of sulphate formation will probably be greatest during the first few seconds following emission before substantial dilution of the plume can occur. Kittelson *et al* (1999) made an extensive study of secondary particle formation in diesel exhausts that suggested that secondary particles can account for up to 14% of total particle emissions. Possibly as much as 10% of the sulphur dioxide may be converted to sulphuric acid during the first few seconds following emission. Subsequent rates of conversion of SO₂ to sulphate are likely to be low compared with the rates at which pollutants are normally transported within the atmosphere. Only a small percentage (<5%) is likely to be converted to sulphate within 10 km of emission (Gupta *et al*, 1995; Lazaridis and Melas, 1998). The contribution to secondary particle concentrations at greater distances from the plant would be immeasurably small compared the concentrations of secondary particles likely to arise as a result of emissions from other sources. The overall increment in sulphate concentrations as a result of the formation of secondary particles would be very small, perhaps of the order of 5-10 µgm⁻³ in the most heavily impacted areas under the least favourable weather conditions. The effect on annual average concentrations of sulphate particles is likely to be much less than 1 µgm⁻³.

4.8.2 Potential formation of nitrate particles

The emissions of nitrogen oxides from gas fired furnaces are substantial in comparison to those of primary particles. A small component of these nitrogen oxide emissions may contribute to the formation of secondary particles through oxidation to nitric acid vapour by hydroxyl radicals during daytime and to N_2O_5 at night by ozone (QUARG, 1996). Nitric acid does not nucleate as readily as sulphuric acid and is not readily absorbed by particles already containing acidic sulphur compounds (QUARG, 1996). Cloud droplets absorb large amounts of nitric acid but are likely to have a minor role in the formation of particulate nitrate (Kerminen *et al*, 1997). Nitric acid can be absorbed by seasalt, displacing HCl or, in the absence of sulphate, react with NH_3 to form ammonium nitrate that is absorbed by accumulation mode particles (Kleeman and Case, 1998). The rate of conversion of HNO_3 to NO_3^- aerosol is only 7-10%/hour (Lee *et al*, 1999). $NaNO_3$ is relatively non-volatile but NH_4NO_3 may evaporate back to gas phase. N_2O_5 also reacts with sea salt to form sodium nitrate. Rates of NO_x conversion to secondary particles are generally slower than those for the formation of secondary sulphate (QUARG, 1996).

Nitrate particles account for 10-20% of urban particulate by mass and are concentrated in the sub $2.5\mu m$ size range although a larger proportion (30-40% by mass) is found within supermicron sized particles than for sulphate (COMEAP, 1995). This is due to absorption and reaction with of nitrogen oxide species with sea salt and other particles and supermicron particles are a significant sink for nitrate (Kerminen *et al*, 1997). Nitrate concentrations have generally increased since measurements started in Harwell in 1954.

The contribution of NO_x emissions to the formation of secondary particles will be small in comparison to the secondary particle formation associated with sulphur dioxide. Other potential effects of NO_x emissions on atmospheric chemistry include the formation of acid rain and reactions leading to the accumulation of ozone.

4.9 IMPACT OF GASEOUS EMISSIONS ON LOCAL AIR QUALITY

The particle emissions from glass furnaces only form a small proportion of total emissions (Table 3.5) and are likely to have a very small effect on long term mean concentrations of airborne particulate. Emissions of sulphur and nitrogen oxides are also unlikely to have a measurable effect on long term mean concentrations of sulphur dioxide or nitrogen dioxide in local air (Table 4.8). Emissions of sulphur and nitrogen oxides are, however, likely to add substantially to local concentrations of sulphur dioxide and nitrogen dioxide during periods of relatively poor dispersion. The dispersion modelling results suggest that the 1 hour air quality objective for nitrogen dioxide may be exceeded on rare occasions as a result of emissions from glassworks (UK5, UK6) in St. Helens (Table 4.8). At present 18 exceedences are allowed per annum and it is highly unlikely that emissions from the glassworks would lead to that number of exceedences of the standard in any year.

Table 4.8 Predicted increments in concentrations of gaseous pollution associated with emissions from a gas-fired glass furnace (μmg^{-3}) as estimated from emission data and dispersion modelling results provided by Pilkington. The UK government's air quality objectives are shown for comparison.

	1 hour		annual		Background	Air quality objective		
	UK5	UK6	UK5	UK6		1 hour	24 hour	annual
SO ₂	75	39	0.5	0.3	2.1-4.0	350 (266*)	125	20
HCl	2.7	1.5	0.01	0.01				
HF	0.46	0.26	0.003	0.002				
NO _x	238	144	1.6	1.2	15.1-20	200		40

**15 minute mean*

The model results suggest that gaseous emissions are more likely to contribute to exceedences of the Government's Air Quality Objectives than particle emissions.

4.10 OVERALL IMPACT OF PARTICLE EMISSIONS FROM GLASS FURNACES ON LOCAL AIR QUALITY

The emissions from Pilkington's glassworks in St Helens have a small effect on local air quality. They do not contribute to measurable increments in annual concentrations of PM₁₀, sulphur dioxide or NO_x, even in the most heavily impacted areas. The emissions could, however, contribute to measurable increments in concentrations of these pollutants during episodes of poor dispersion. It is very unlikely that emissions will lead to exceedences of the Government's current air quality objectives in the surrounding area. The formation of secondary particles, particularly sulphuric acid, will have a small long term effect on local air quality.

5. HEALTH EFFECTS ASSOCIATED WITH INSOLUBLE PARTICLE EMISSIONS FROM GAS-FIRED GLASS FURNACES

5.1 INTRODUCTION

This chapter discusses the health effects that could potentially be associated with the main components of the insoluble fraction of particle emissions from glassworks. These include heavy metals and crystalline silica which are both associated with adverse effects at high levels of exposure.

5.2 HEAVY METALS

5.2.1 General

The increments in heavy metal pollution are generally extremely small compared with ambient concentrations in UK air (Tables 4.6-4.8). Predicted levels are well below relevant exposure standards (Table 5.1) and are insignificant in comparison to the levels of exposure associated with toxicity. The toxicity of heavy metals varies both with the type of metal and also between different compounds of an individual metal. The bioavailability of inhaled metals plays a key role in determining their toxicity. Generally bioavailability is related to solubility and for most metals and metal compounds toxicity increases with increasing solubility reflecting increased levels of absorption, although occasionally increasing solubility may be linked to increased levels of excretion. Absorption of soluble metal species into the blood stream from the lung can be very rapid and metal toxicity can be greater following inhalation than other routes of exposure. The biological activity of many metals is related to their ability to occur in several oxidation states (Nemery, 1990; Stohs and Bagchi, 1995). This enables them to take part in Fenton redox cycles leading to the release of reactive oxygen species causing tissue damage and a further inflammatory response (Stohs and Bagchi, 1995). Some metals exert their toxic effects by substituting for iron within the many enzymes that govern body chemistry leading to an impairment of enzyme activity. Some heavy metals are associated with various respiratory effects including lung cancer, but others are more strongly associated with liver, kidney damage or neurotoxicity. In general terms the main route of exposure to most heavy metals is from drinking water rather than air pollution. Significant exposures to some heavy metals may occur around metal processing plants but airborne concentrations greatly exceed those likely to be associated with glass furnaces. Summaries of the health effects associated with the specific heavy metals in glass furnaces emissions based on the relevant publications of the WHO (1981, 1988a, 1988b, 1995, 1998) are given below.

Table 5.1 Comparison of predicted levels of heavy metal pollution with relevant exposure limits

Metal	Maximum increment ngm^{-3}	WHO-Europe		EPAQS	OEL/100 ngm^{-3} ***
		limit	lifetime risk of cancer/ ngm^{-3}		
Chromium	4.0		$(4 \times 10^{-5})^{**}$		5000
Iron	1.5				20000
Copper	0.4				20000
Lead	0.6	500		250	1500
Arsenic	0.2-1.5		1.5×10^{-6}		1000
Vanadium*	<0.2				

**only present in emissions from furnaces where oil is burnt*

***as Cr (VI) – note that CR (VI) has not been detected in glassworks emissions by Pilkington*

**** Occupational exposure limit/100 – allows for the difference in time spent at work (20% of time during working lifetime, or approximately 10% of total lifetime) and incorporates a margin of uncertainty to allow for the presence of susceptible individuals in the general population who would not be present in the workplace.*

5.2.2 Effects associated with specific metals in emissions

Chromium is an essential nutrient although excessive occupational exposure to airborne chromium can cause adverse effects. These include irritation of the upper respiratory tract, allergic reactions and cancers of the respiratory tract. Chrome (VI) is a lung carcinogen, but there is no evidence to link the lower oxidation states of chrome, such as are present in emissions from glass furnaces, with cancer. There is no evidence of any lung cancer excess associated with environmental exposure to chromium concentrations of 1000ngm^{-3} in the vicinity of ferro-alloy plants. The much lower concentrations of chromium associated with glass furnace emissions do not represent a measurable risk to health.

Lead interferes with a wide range of physiological processes and children are particularly susceptible to the effects of lead poisoning. Dose-response information about the effects of lead is generally given in terms of blood lead levels. The relationship between concentrations of airborne lead and blood lead levels is complex, but an increase of 1000ng/m^3 in airborne lead concentrations is associated with an approximate $5 \mu\text{g/dl}$ increase in blood lead concentrations. This suggests that the increment in lead concentrations from the furnace will have a negligible effect on blood lead levels in the local population. The air quality standard set by EPAQS (1998) is intended to prevent impairment of brain development due to lead exposure. Subtle effects on IQ have been found at relatively low levels of lead exposure ($10 \mu\text{g/dl}$ in blood). An increase in 10 to $20 \mu\text{g/dl}$ is associated with a reduction in average IQ of about 2 points. At higher levels of exposure ($50 \mu\text{g/dl}$) anaemia can arise and at still higher levels stomach cramps. Concentrations in blood exceeding $100 \mu\text{g/dl}$ are associated with brain damage and damage to other organs such as the kidney. The main route of exposure is normally through drinking water and the uptake of lead from air pollution is normally tiny in comparison with that from water and food. The increment in airborne lead concentrations associated with glass making is small compared with the air quality standard recommended by EPAQs. It would not be expected to result in a detectable increase in risk to the health of young children.

The main route of human exposure to arsenic is through drinking water and food, particularly sea food and arsenic poisoning is generally associated with ingestion rather than inhalation

exposure. Adverse effects are associated with a daily oral intake of 100-200µg and the WHO recommend that daily intake is restricted to less than 2µg. This would be equivalent to exposure to 67ngm⁻³ for an adult inhaling 30m³ of air over a 24 hour period assuming similar efficiencies of absorption as for the ingested dose. Occupational exposure to airborne arsenic is associated with conjunctivitis, perforation of the nasal septum as a result of irritation of the upper respiratory tract and lung cancer. The additional risk of lung cancer likely to be associated with arsenic emissions from glass furnaces in St Helens is less than 2 in a million for people living in the most heavily impacted area. Overall the effects of arsenic would be expected to be immeasurably small.

The main route of exposure to vanadium is often food although only about 0.1-1% of soluble vanadium is absorbed from the human gastrointestinal tract. In contrast about 25% of inhaled vanadium in soluble salts is absorbed. Inhaled vanadium is largely cleared from the lungs within the first three days following exposure. It has been suggested that current levels of vanadium in ambient air have been associated with mortality from heart and lung disease, but the results of available studies are equivocal. In occupational settings long term exposure to 10 000 – 40 000 ngm⁻³ has been associated with irritation but not with any reduction in lung function. Atopic rhinitis, bronchitis and nonspecific effects such as headache, nausea, weakness, ringing in the ears and palpitation are associated with considerably higher levels of exposure to vanadium. Levels of emissions of vanadium from glassworks are too small to give rise to measurable effects.

Both iron and copper are essential nutrients and would only be associated with toxic effects following excessive levels of exposure. Both elements have multiple oxidation states and can take part in redox reactions. Their presence in air may contribute to the toxic effects of PM₁₀, although their intrinsic toxicity is very low. Short term exposure to very high concentrations of iron oxide or copper fume may cause metal fume fever giving rise to flu-like symptoms including fever that are normally reversible, but can be fatal. Emissions of iron and copper from glass furnaces are not likely to have a directly measurable effect on health.

The trace amounts of other metals that are reported in glass furnace particles are also emitted to the atmosphere in immeasurably small quantities and will not have a significant effect on health.

5.2.3 Heavy metals in PM₁₀

Heavy metals have been implicated in the toxicity of PM₁₀ despite being present at very low concentrations in urban areas (Donaldson *et al*, 1997). Bioavailability may play a key role in governing toxicity as metals bound to the surface of other types of particle such as diesel soot are readily taken into solution (Dreher *et al*, 1997). A number of experiments have demonstrated the toxicity of the soluble metals content of PM₁₀ (Gilmour *et al*, 1996, Carter *et al*, 1997, Quay *et al*, 1998). The metals content of emissions from glassworks is generally less in terms of mg/kg than for urban PM₁₀ in general with the possible exception of chromium. The absolute quantities of heavy metals in air pollution are, however, likely to be less important than their bioavailability when inhaled. Any metals within the soluble sodium sulphate component of emissions are likely to be taken rapidly into the blood stream. The analytical data, however, suggests that most of the metals content is in the insoluble fraction, probably incorporated within glass beads. These glass beads have a relatively low alkali content and comparison with glass fibres suggests that it would probably take several months for a glass bead to disappear by dissolution (Searl and Buchanan, 2000). The glass beads are likely to be cleared by cellular processes before extensive leaching of metals can occur and the metals are therefore likely to be less bioavailable than those absorbed onto the surface of urban soot particles.

5.2.4 Conclusions

The contribution of heavy metal emissions from glass furnaces is tiny in comparison to emissions from transport, power generation and metals industry (see QUARG, 1996). The increment in local concentrations of heavy metals due to emissions from glassworks is far too small to have a measurable impact on human health. Much of the heavy metals content of emissions is likely to be contained within insoluble silicate glass particles and have a relatively low bioavailability when compared with metals adsorbed onto soot particles from other sources. Even allowing for potential additive effects of different metals and the absorption of metals onto other particles, the heavy metals in emissions from glass furnaces are insignificant in terms of risks to the health of local affected communities.

5.3 CRYSTALLINE SILICA

Crystalline silica is an abundant component of soils and rocks and is normally present in small concentrations in PM₁₀. The increment in concentrations of crystalline silica due to emissions from glass furnaces is negligible and total concentrations of crystalline silica in ambient air are less than about 2µgm⁻³. The risks of developing mild silicosis as a result of 40 years workplace exposure to 100µgm⁻³ during working hours are less than 1%. The overall risks to health associated with crystalline silica emissions from glass furnaces would be insignificant.

5.4 OTHER MINERAL SPECIES

Most of the other minerals identified in emissions are not specifically associated with toxic effects and all of these species would be present in the plume at extremely low concentrations relative to those likely to cause adverse effects. Of the named mineral species that have been found, only cassiterite and fluoride would be subject to specific limits on occupational exposure (for tin and for fluoride).

6. SODIUM SULPHATE TOXICITY: ANIMAL STUDIES

6.1 INTRODUCTION

This chapter examines the evidence from animal experiments for toxic effects associated with exposure to sodium sulphate. The first part of this chapter outlines the information about sodium sulphate toxicity that is available from the RTECS database. This information is not specific to inhalation and cannot be used to assess the effects of long term inhalation exposure to low concentrations. There have been very few studies relevant to the inhalation toxicology of sodium sulphate and there is considerably more information available about other sulphate species such as are found as secondary particles in urban air. The main part of the chapter therefore considers sulphate species more generally. The conclusions drawn at the end of the chapter consider the likely effects on health associated with sodium sulphate and secondary sulphate species from glass furnace emissions as assessed from the animal data.

6.2 SODIUM SULPHATE TOXICITY

There is relatively little toxicity data reported for sodium sulphate in the Registry of Toxic Effects of Chemical Substances (RTECS) database. This is presumably because there has been inadequate suspicion of human health effects to justify an extensive study. Sodium sulphate has a low acute toxicity (Table 6.1) but may have adverse effects on developing foetuses at moderate levels of exposure (Table 6.2). The toxic effects of sodium sulphate appear to be much greater following intravenous or parenteral administration than following oral administration (Tables 6.1, 6.2). The high solubility of sodium sulphate means that it is unlikely to be persistent in lung tissue and suggests that it is less likely to be associated with a persistent inflammatory response than more persistent materials. The high solubility of sodium sulphate also means that any toxic effects are more likely to be associated with an excess of sodium or sulphate in body fluids than specifically with sodium sulphate.

Table 6.1 Acute toxicity data for sodium sulphate (from RTECS): LD₅₀ – dose at which 50% of experimental animals perish; LD_{LO} – lowest dose at which lethal effects have been reported

Species	Route of Administration	LD ₅₀ mg/kg	LD _{LO} mg/kg	Effects
mouse	oral	5989		not reported
rabbit	intravenous	1220		not reported
mouse	intravenous		1220	not reported

Table 6.2 Reproductive toxicity of sodium sulphate (from RTECS)

Species	Route of Administration	Timing of administration	TD _{LO} mg/kg	Effects
mouse	oral	8 days after conception	14000	effects on newborn
mouse	parenteral	8 days after conception	60	fetotoxicity (excluding death)

Both sodium and sulphate are normally present in body fluids but the administration of excessive quantities of either ionic species can have adverse effects. The sulphate ion does not cross cell membranes readily and oral administration as sodium sulphate tends to add water to the bowel and cause diarrhoea. Parenteral administration of sodium sulphate tends to lead to an

increased volume of urine due to the excretion of salt and water. In animal experiments this has been shown to cause calcium deficiency and affect the serum levels of potassium, sodium and other electrolytes. Overall, however, sodium sulphate would only be expected to cause adverse health effects at extremely high levels of exposure, several orders of magnitude greater than might result from exposure to emissions from glass furnaces.

6.3 INHALATION TOXICOLOGY OF SOLUBLE SULPHATE SPECIES

6.3.1 Irritancy and bronchial responsiveness

Exposure to particles, SO₂ and NO_x in urban air is associated with the exacerbation of asthmatic symptoms. There is limited experimental evidence to suggest an association between bronchial responsiveness and exposure to airborne sulphate, particularly as sulphuric acid. El Fawal and Schlesinger (1994) found that a single 3 hour exposure of rabbits to 75 µgm⁻³ of sulphuric acid induces increased bronchial hyper-responsiveness. Kitabatke *et al* (1991) used a guinea pig model to show that exposure to concentrations of 200-2000 µgm⁻³ ammonium sulphate can cause asthmatic dyspnoea and increase sensitivity to acetylcholine. Bronchoconstriction has been reported in guinea pigs following exposures to sulphuric acid concentrations that are only a few times higher (75µgm⁻³) than typical of ambient air and within the range of concentrations found during high pollution events (Amdur, 1996).

Amdur *et al* (1978) found that the irritant effect of sulphate salts (as submicron particles) in animal experiments was governed by their cation content with all the salts tested being less potent than sulphuric acid (Table 6.3). The incorporation of any transition metals into sulphate salts or adsorption of metals on particle surfaces might therefore be expected to have a critical effect on their toxic potential. Sodium sulphate was a much less potent irritant than the other sulphates tested (Table 6.3), but its potency in glass furnace emissions might be slightly raised by the absorption of acidic species and other metals on particle surfaces. Amdur *et al* (1978) also established that the potential of sulphate salts to cause irritancy was not directly related to their potential to cause adverse effects. Cupric sulphate had the lowest irritant potency of the salts tested but was the only salt that potentiated the effects of sulphur dioxide (SO₂). Irritation of the bronchial airways gives rise to the release of histamine that in turn gives rise to inflammation and potential narrowing of the airways. Charles and Menzel (1975) investigated the release of histamine from guinea pig lung fragments and found that ammonium sulphate was more potent at inducing histamine release than ammonium acetate or nitrate. This could, however, simply reflect the greater acidity of ammonium sulphate. More recent studies have confirmed the importance of transition metals such as V, Fe and Ni rather than sulphate in driving adverse effects (eg Gavett *et al*, 1997).

Table 6.3 Relative potency of sulphate compounds to cause irritancy in animal experiments (from Amdur *et al*, 1978)

Compound	Relative potency
Sulphuric acid	100
Zinc ammonium sulphate	33
Ferric sulphate	26
zinc sulphate	19
ammonium sulphate	10
ammonium bisulphate	3
cupric sulphate	2
sodium sulphate	0.7

Overall, the animal data suggest that sulphate compounds can induce irritancy and bronchial responsiveness in animals exposed to concentrations that are considerably greater than those typical of ambient air. The relative potency of sulphate compounds to cause effects is related to their acidity and also to their transition metals content. Sodium sulphate has a much lower potential to cause irritation and bronchial hyper-responsiveness following inhalation than other sulphate compounds.

6.3.2 Lung function and cardiac effects

Exposure to ambient air pollution is associated with both short and longer term detrimental effects on lung function and there has been some experimental investigation of the role of different components of particulate pollution in causing adverse effects. Sackner *et al* (1981) examined the effects of short term (4 hour) exposures to submicron aerosols of NaCl, Na₂SO₄, NH₄HSO₄, (NH₄)₂SO₄, ZnSO₄, ZnNH₄SO₄, Al₂(SO₄)₃, MnSO₄, NiSO₄, CuSO₄, Fe SO₄ and Fe₂(SO₄)₃ on lung function of anaesthetised dogs. They found no changes in lung function at doses of 4100-8000µgm⁻³. One mg intravenous injections of these compounds had no effects on lung function or cardiac function. 100mg injections however of ZnSO₄, ZnNH₄SO₄ induced a fall in cardiac output, systemic hypotension, hypoxemia and metabolic acidosis. Copper sulphate produced pulmonary hypertension, fall in cardiac output, hypoxemia, respiratory acidosis and decrease of specific total respiratory conductance. These adverse effects appear to have been associated with transition metal cations rather than with sulphate. Longer term experiments with ammonium sulphate have demonstrated that 4 weeks exposure to 1000 µgm⁻³ (30 hours/week) can aggravate the effects of elastase-induced emphysema in rats as assessed from changes in lung morphology and lung function (Loscutoff *et al*, 1985; Busch *et al*, 1984). Similar effects were not, however, observed in guinea pigs.

Pepelko *et al* (1980) found that exposure of rats to 300 000µgm⁻³ of ammonium sulphate over 14 days had no effect on respiratory parameters such as respiratory volume, vital capacity and blood gases. In contrast, exposure of guinea pigs to 800 000 – 900 000µgm⁻³ caused death in 40% of the test animals. Exposure of donkeys to sulphuric acid aerosols (Mass Median Aerodynamic Diameter, MMAD, 0.4µm) at 1000-1500µgm⁻³ or ammonium sulphate (MMAD 0.4µm) 2000µgm⁻³ had no significant effect on lung function (Schlesinger *et al*, 1978). Long term exposure of monkeys to sulphuric acid mist (400µgm⁻³) is associated with a deterioration in lung function and changes in lung structure including epithelial hyperplasia and thickening of the bronchiolar walls (Alarie *et al*, 1975; 1973).

Overall, effects appear to be largely associated with relatively high concentrations of acidity and with transition metals. There is no evidence to suggest that exposure to small concentrations of sodium sulphate is likely to have an adverse effect on lung or cardiac function.

6.3.3 Clearance

The effective clearance of foreign bodies from the airways plays an important role in the maintenance of respiratory health. Schlesinger (1983) examined the effects of 1 hour exposures to submicron aerosols of ammonium sulphate, ammonium bisulphate and sodium sulphate on mucociliary clearance in rabbits. He found that the effects were least with sodium sulphate and none of the salts had a significant effect at exposures of less than 2000µgm⁻³. Subsequently Schlesinger (1989) found that exposure of rabbits to concentrations of 1000µgm⁻³ sulphuric acid (MMAD 0.3µm) was associated with a doubling in clearance time. The effects of soluble sulphates in causing retardation of lung clearance increased with increasing acidity with sodium sulphate having the smallest effect and sulphuric acid the greatest effect (H₂SO₄>HN₄HSO₄>(NH₄)₂SO₄>Na₂SO₄). The concentration required to cause

an effect on clearance decreased with increasing length of exposure time in approximate proportion to the relative length of time. Gearhart and Schlesinger (1988) found that chronic exposure of rabbits to $250\mu\text{gm}^{-3}$ ($0.3\mu\text{m}$) of sulphuric acid for 1 hour/day on 5 days/week was associated with impairment of tracheobronchial mucociliary clearance. This impairment worsened with continued exposure and did not recover after the end of exposure. After 4 months of exposure animals developed airway hyperresponsiveness which worsened by 8 months exposure but thereafter appeared to stabilise. Both bronchial sensitivity and bronchial reactivity were substantially different from that in control animals. The exposure had little effect on lung function before 12 months of exposure. The extrapolation from the hourly exposures to 24 hour 7 day week exposure is unclear. If a simple time concentration factor is used, this would be equivalent to a continuous exposure to $15\mu\text{gm}^{-3}$ H_2SO_4 . These levels are slightly higher than typical environmental concentrations of sulphuric acid in the UK and considerably higher than would be expected to be associated with emissions from glass making by Pilkington in St. Helens.

Rabbits appear to be relatively sensitive to aerosol acidity and clearance in other species is affected to varying degrees by aerosol acidity. Phalen *et al* (1980) established that exposure of rats to $3600\mu\text{gm}^{-3}$ of ammonium sulphate had no effect on clearance. Similarly, a four hour inhalation exposure to $5000\mu\text{gm}^{-3}$ sodium sulphate did not change tracheal mucous velocity in sheep (Sackner *et al*, 1981). In contrast, Phalen *et al* (1980) found that short term exposure of rats to $3600\mu\text{gm}^{-3}$ ferrous sulphate in a dry atmosphere caused an acceleration of macrophage-mediated clearance but exposure to the same concentration in combination with 0.8ppm ozone had no effect. Chronic inhalation of sulphuric acid (H_2SO_4) ($0.4\mu\text{m}$) by donkeys at concentrations greater than $200\mu\text{gm}^{-3}$ has a similar effect on clearance mechanisms to heavy smoking (Schlesinger *et al*, 1978). In some donkeys, the retardation of clearance continued beyond the cessation of exposure (Schlesinger *et al*, 1978). The effects of the acid exposures were greatest between 30 and 60 minutes after the initiation of exposure suggesting that the acid takes a time to produce an effect. Effects appeared to be associated with acidity rather than sulphate as exposures to concentrations of up to $30000\mu\text{gm}^{-3}$ of ammonium sulphate had no effect on clearance (Schlesinger *et al*, 1978).

Overall the potency of sulphate compounds to have an adverse effect on clearance appears to be linked to their acidity. Long term exposure to relatively low concentrations of acidity may have a greater long term effect on clearance than single short term exposures to much higher levels of acidity. There is limited evidence that exposure to transition metal sulphates may also affect clearance mechanisms. There is no evidence to suggest that exposure to low concentrations of sodium sulphate would have an adverse effect on clearance.

6.3.4 Macrophage function

Pulmonary macrophages have an important role in clearing foreign bodies from the lung and in the maintenance of respiratory health. One potential way in which exposure to air pollution may lead to adverse health effects is in reducing resistance to infectious respiratory illnesses. Chen *et al* (1992a) found changes in macrophage activity in guinea pigs following exposure to $350\mu\text{gm}^{-3}$ sulphuric acid. Ultrafine sulphuric acid ($0.04\mu\text{m}$ MMAD) depressed activity whereas fine ($0.27\mu\text{m}$ MMAD) increased phagocytic activity. The particle size would have been important in determining the site of deposition within the lung but also in determining the surface area per unit mass. Schlesinger *et al* (1992) found that exposure to 75 or $125\mu\text{gm}^{-3}$ of sulphuric acid depressed macrophage activity in rabbits whereas exposure to $50\mu\text{gm}^{-3}$ did not. The effects included depression in superoxide production and the production of tumour necrosis factor. The exposures did not have an effect on cell numbers or viability and there were no indications of cell injury as assessed from the concentration of lactate dehydrogenase

in lavage fluids. Short term exposure of hamsters to $48000\mu\text{gm}^{-3}$ copper sulphate, $3100\mu\text{gm}^{-3}$ zinc sulphate or $7800\mu\text{gm}^{-3}$ ferrous sulphate has been found to rise to a short term decrease in the phagocytic activity of macrophages (Skornik and Brain, 1983). Similarly, Drummond *et al* (1986) found that short term exposure to $2540\mu\text{gm}^{-3}$ copper sulphate led to a significant reduction in pulmonary bactericidal activity in mice and repeated short exposures to $100\mu\text{gm}^{-3}$ were associated with an increased mortality rate following exposure to *Streptococcus-zooepidemicus*. In contrast exposure to $2710\mu\text{gm}^{-3}$ of ammonium sulphate was associated with significantly increased bactericidal activity. Zelikoff *et al* (1994) demonstrated in an *in vivo* study that the repeated inhalation of sulphuric acid is likely to reduce the uptake and intracellular killing of pathogenic bacteria by exposed pulmonary macrophages. Exposure to sulphuric acid also depresses the production of important biological modifiers that are critical for maintaining pulmonary immunocompetence. Zelikoff *et al* (1994) suggest that this may be relevant to understanding the association between exposure to sulphate and increased incidence of acute bronchitis and lower respiratory illness in school-age children. None of these experimental results suggest that exposure to low concentrations of sodium sulphate would be expected to have an adverse effect on macrophage function.

6.3.5 Species differences in sensitivity to sulphate species

There is considerable interspecies variation in sensitivity to sulphuric acid. Rats and monkeys are relatively insensitive to the effects of sulphuric acid whereas mice and guinea pigs are much more susceptible (Last, 1991). Schlesinger *et al* (1992) concluded that sulphuric acid had a much greater effect on phagocytic activity in guinea pigs than in other species with an overall ranking of guinea pig > rat > rabbit > human. The relative sensitivity of different species may vary with the endpoint examined. Loscutoff *et al* (1985), for example, found that $(\text{NH}_4)_2\text{NaSO}_4$ had a much greater effect on pulmonary function in elastase-treated rats than in similarly treated guinea pigs. The pattern of deposition of a sulphate salt in the airways may differ considerably between species because of the effects of hygroscopic growth of particles within the airways. The extent to which acid aerosols may be neutralised within the airways is also likely to vary between species.

6.3.6 Particle size effects

The particle size of sulphate aerosols has an important influence on their deposition within the respiratory tract that in turn affects their potential to cause adverse effects. The response of guinea pigs to the effects of acid aerosol is closely linked with particle size. Particles with a mass median diameter of $7\mu\text{m}$ have little effect on airway resistance probably because they are trapped in the upper airways. Particles with a median diameter of $1\mu\text{m}$ produce a swift response because of their much greater penetration of the lung. Amdur *et al* (1978) found some evidence that, for a given mass concentration, the irritant potential of sulphate salts increased as particle size decreased. This could reflect differences in deposition, but might also reflect the importance of particle surface area in determining effects.

The particle size of sulphate aerosols is likely to be modified subsequent to inhalation through the absorption of moisture from the airways. The high water solubility of sulphuric acid, for example, means that the aerosol may preferentially dissolve in the upper airways with very little reaching the deep lung (Last, 1991). The initial size of aerosol particles has a crucial effect on determining their potential for growth within the respiratory tract. This suggests that there is likely to be a complex relationship between particle size and deposition pattern and ultimate effects.

6.3.7 Mixed exposures

The emissions from Pilkington's plants will mix with a range of other pollutants and the effects of exposure to the mixture may be different from those directly associated with emissions from Pilkington's plants. There has been some experimental interest in mixed exposures to particles, particularly acid aerosols and ozone as occur during summertime photochemical pollution events. COMEAP (1995) reviewed a number of animal studies in which exposure to a range of particulate materials including ammonium sulphate, sulphuric acid, soot and carbon black, increased airways reactivity to ozone and other oxidants. Exposure of rats to ultrafine (0.03 μm) sulphuric acid, for example, potentiates the response of rat lungs to ozone as assessed from increased protein content and apparent increased collagen synthesis (Last, 1991). Co-exposure to ozone and aerosols of neutral salts such as sodium sulphate or chloride does not however, give rise to synergistic effects. The acidity of the aerosol, rather than sulphate content potentiates the effects ozone (Last *et al*, 1984). Last *et al* (1984) also demonstrated that particle size was important as effects were seen with 1 $\mu\text{g}\text{m}^{-3}$ of a 0.5 μm aerosol but not with 0.02 μm particles. This may relate to the site of deposition relative to the penetration of ozone into the airways and also to the quantity of acid deposited over a given area of the airways (COMEAP, 1995). Chen *et al* (1992b) also demonstrated a significant interaction between ozone and sulphuric acid, although the effects of the ozone were to counteract those of sulphuric acid when both were administered together. The exposure of rabbits to 125 $\mu\text{g}\text{m}^{-3}$ sulphuric acid reduced the intracellular pH of pulmonary macrophages as did the exposure of rabbits to 0.1-0.6ppm ozone. Exposure to 50 $\mu\text{g}\text{m}^{-3}$ acid and 0.6ppm ozone separately did not significantly affect macrophage pH whereas the mixture produced a significant interaction.

The condensation of sulphuric acid from oxidation of sulphur dioxide emissions from Pilkington's plants may lead to the formation of relatively complex metal-acid particles. Sulphuric acid in ambient air is sorbed on ultrafine metal oxides and animal experiments have shown that sorbed sulphuric acid is a more effective irritant than free sulphuric acid (Amdur, 1996). Guinea pig lungs respond to 30 $\mu\text{g}\text{m}^{-3}$ of sulphuric acid sorbed on fine particles, show cumulative dose related responses to repeated exposures and increased sensitivity to further exposure to sorbed sulphuric acid or ozone (Amdur, 1996). Jakab *et al* (1996) created an aerosol of sulphuric acid coated carbon black by lofting carbon black in the presence of sulphur dioxide under conditions of relatively high humidity. Exposure of rats to 10 000 $\mu\text{g}\text{m}^{-3}$ of this aerosol caused a decrease in the phagocytic activity of macrophages not seen in rats exposed to the same concentration of uncoated carbon black. The insoluble metal and soot component of emissions from glass furnaces is extremely small, so this type of effect is probably of limited importance in terms of the particle emissions. Sulphur dioxide emissions from glass furnaces, however, may react with metal and soot particles from other sources and contribute to adverse effects. The concentrations of absorbed sulphur species are likely to be much smaller than those associated with adverse effects in experimental systems.

Overall, the limited experimental data available suggests that the effects of inhaled sulphate species, especially sulphuric acid may be substantially modified by concurrent exposure to other air pollutants.

6.3.8 Inhalation toxicology of sulphite

Guinea pigs and dogs exposed to sulphite aerosols show adverse respiratory effects and sulphite is generally considered to be more biologically active than sulphate (Amdur, 1996). Sulphite has not been detected within emissions from glassworks, but may form from SO₂ emissions as a secondary pollutant in the atmosphere.

6.4 POSSIBLE *IN VITRO* INVESTIGATION OF WASTE GAS DUST FROM GLASSWORKS

There is a paucity of published studies on the toxicity of sodium sulphate and some uncertainty over the potential toxicity of the emissions from gas plant flues. In particular, it is possible that trace components of the waste gas dust may greatly affect its toxicity relative to that expected for pure sodium sulphate. It would be possible to investigate the toxicity of the waste gas dust further in a series of *in vitro* assays relevant to inhalation effects. The toxic effects of the waste gas dust could be compared with those of a purer form of sodium sulphate and with particulate materials for which there is good information about their inhalation toxicology such as DQ12 Quartz (as a positive control) and titanium dioxide (as a relatively inert control). In addition, it might be instructive to compare the waste gas dust with a sample of more typical ambient urban PM₁₀, although interpretation of the results could be difficult because little is known about the relative toxicity of PM₁₀ in different urban areas. It would also be possible to compare the waste gas dust with ammonium sulphate, the main sulphate species present in typical urban air.

Assays could be selected to investigate the effects of the dusts on various aspects of cell function for different types of target cell within the respiratory tract such as alveolar macrophages, bronchial epithelial cells and alveolar type II epithelial cells. One suitable measure of particle toxicity that has been widely used in *in vitro* tests is the release from particulate-exposed cells of the enzyme lactate dehydrogenase (LDH), a marker of cell injury and death (Donaldson *et al.*, 1988; HSE, 1999).

6.5 CONCLUSIONS

Overall, the results of animal experiments suggest that the inhalation of sulphate itself is not specifically associated with adverse effects, even at concentrations that are hundreds or even thousands of times greater than those found in ambient air. Animal experiments with pH neutral or near neutral sulphate salts generally do not produce adverse respiratory effects in animals (Lippmann and Thurston, 1996). The toxicological information available from animal studies suggests that short term exposure to the predicted maximum concentrations of sodium sulphate (<100µgm⁻³) would not be expected to lead to any adverse effects.

The inhalation of acidic sulphate species, particularly sulphuric acid, and some transition metal sulphates, however, can cause adverse effects in animals at much lower concentrations than for sodium sulphate (Amdur, 1996; MAAPE, 1992). This is of particular relevance to the potential formation of secondary sulphuric acid particles associated with emissions from glass furnaces. The particle size of any sulphuric acid/ sodium sulphate droplets/particles associated with emissions from glass furnaces is likely to be somewhat greater than has typically been used in animal experiments. This suggests that higher concentrations of these particles might be tolerated, even in sensitive species because of low levels of deposition in the lower airways. The increments in sulphuric acid concentration are likely to be a tiny fraction of those associated with longer term effects on health in monkeys (1000 µgm⁻³). Overall, the results of animal experiments suggest that exposures to low concentrations of acid aerosol associated with sodium sulphate emissions are unlikely to be associated with adverse health effects in most individuals.

7. SODIUM SULPHATE TOXICITY: HUMAN VOLUNTEER STUDIES

7.1 INTRODUCTION

The use of human volunteers in experimental exposures to air pollutants allows investigation of the specific effects associated with individual pollutants or with simple mixtures of pollutants. Human volunteer studies, however, do not include individuals with severe asthma or otherwise compromised health and may be of limited value for the prediction of health effects in the wider community (MAAPE, 1992). Volunteer studies necessarily involve only short term exposure and are not informative about potential long term effects. Relatively few experiments have been conducted with sulphate salts or sulphuric acid although the interest in the potential association between acid aerosols and health during the 1980s led to a few experimental studies that are reviewed in this chapter. The hygroscopic nature of many soluble sulphate species may have a substantial effect on the size of inhaled particles and their deposition within the respiratory tract. This in turn may affect their potential effects on the respiratory system. The initial part of this chapter therefore considers the behaviour of soluble particles within the respiratory tract. The investigations of soluble particle behaviour in the respiratory tract have used mainly casts of human respiratory systems and mathematical modelling rather than human volunteers, but the results provide a useful background to the exposure studies. Few of the volunteer studies specifically address the issue of hygroscopic particle growth within the respiratory tract. Given the paucity of information about sodium sulphate specifically, the effects of other neutral sodium salts including sodium chloride and sodium nitrate have also been reviewed.

7.2 FATE OF SOLUBLE PARTICLES WITHIN THE RESPIRATORY TRACT

The relative humidity of the human respiratory tract is normally greater than that of inhaled air which is rapidly humidified as it passes through the respiratory tract (Sarangapani and Wexler, 1996). One consequence of this humidity is that any hygroscopic particles that are inhaled will tend to absorb water and grow in size. Particle growth within the airways is size dependent with the growth of submicron diameter particles being particularly rapid (Ferron, 1977; Cocks and Fernando, 1982). The absorption of water by inhaled particles of a soluble salt has been shown to substantially alter the pattern of deposition in comparison to similar particles of an insoluble material (Ferron, 1977; Ferron *et al*, 1983). The proportion of particles greater than 2 μm of an insoluble material deposited in the nose and throat is about 30% and few particles less than 1 μm are deposited in this region. In contrast the proportion of soluble particles greater than 2 μm deposited in the nose and throat ranges is greater than 50%. The cut off for deposition of soluble particles in this region is 0.5 rather than 1 μm . A much lower proportion of soluble particles reach the deep lung than for an insoluble aerosol of comparable size characteristics. While this phenomenon will not affect total inhaled dose, it could lead to a relatively greater deposition within the upper airways and a much reduced level of deposition in the deep lung compared with other components of PM₁₀. This in turn might influence the type of health effects that may develop as a result of exposure to the same nominal dose of soluble PM₁₀ or total PM₁₀. For example, soluble particles may be more likely to be associated with upper airways symptoms than effects associated with the deep lung. Smaller particles are more likely to penetrate to the small airways than larger particles and this might be linked to the apparent inverse relationship between airways resistance and particle diameter (MAAPE, 1992). Insoluble particles may carry soluble components deeper into the lung than in the absence of a carrier.

7.3 SULPHATE SALTS

MAAPE (1992) reviewed several studies with ammonium sulphate and ammonium bisulphate that found no consistent effects in healthy volunteers at concentrations of up to $1000\mu\text{gm}^{-3}$. Ambient concentrations of these salts would normally be less than $10\mu\text{gm}^{-3}$ and would rarely exceed $50\mu\text{gm}^{-3}$ (QUARG, 1996). Utell *et al* (1982) found that exposures to $1000\mu\text{gm}^{-3}$ concentrations of ammonium sulphate, sodium bisulphate, ammonium bisulphate and sulphuric acid were associated with a small loss of airways conduction and flow rate, with the greatest effects being asthmatics. No changes were seen following $100\mu\text{gm}^{-3}$ exposures in asthmatics or healthy volunteers. A subsequent more specific study of the effects of acidic sulphate salts (ammonium sulphate, ammonium bisulphate and sodium bisulphate) and sulphuric acid in asthmatic volunteers showed little response at $100\mu\text{gm}^{-3}$ or $450\mu\text{gm}^{-3}$ but measurable responses at $1000\mu\text{gm}^{-3}$ (Utell *et al*, 1984). The extent to which the various sulphate species caused bronchoconstriction was in proportion to their acidity ie $\text{H}_2\text{SO}_4 > \text{NH}_4\text{HSO}_4 > \text{NaHSO}_4 > (\text{HNH}_4)_2\text{SO}_4$. Utell *et al* also found that when sulphate exposure was followed by a carbachol challenge, the bronchoconstrictive action of the carbachol was potentiated by the sulphates in rough relation to their acidity. Kleinman *et al* (1981) exposed human volunteers on two occasions to $75\mu\text{gm}^{-3}$ ferric sulphate. They found no changes in selected lung function parameters but did not investigate bronchial reactivity.

Overall the information from human volunteer studies suggests that aerosol acidity is likely to be far more important than the presence of sulphate in causing adverse respiratory effects. This suggests that effects are unlikely with exposures to $1000\mu\text{gm}^{-3}$ sodium sulphate, given that it will have a much lower acidity than ammonium sulphate or sodium bisulphate. I have not identified any reports of experiments performed specifically with sodium sulphate.

7.4 SULPHURIC ACID AND ACID FOGS

Emissions of both sulphur dioxide and nitrogen dioxide from glass furnaces could potentially contribute to ambient aerosol acidity. The results of human volunteer experiments with sulphuric acid have been highly inconsistent but asthmatic subjects, particularly children and adolescents, appear to be the most susceptible to adverse effects (Table 7.1: MAAPE, 1992; Utell and Samet, 1996). There is some evidence of an association of bronchoconstriction with acidity generally rather than specifically with sulphuric acid. Fine *et al* (1987), for example, found that total titratable acidity was a more relevant measure of exposure than simple pH in human volunteer experiments. The change in S_{raw} associated with hydrochloric acid was only marginally greater than for sulphuric acid, suggesting that the availability of H^+ is more strongly associated with bronchoconstriction than either sulphate or chloride. Similarly Koenig *et al* (1989) found that exposure to similar mass concentrations of sulphuric and nitric acid was associated with similar effects on lung function and airways resistance. Acid fogs may also induce bronchoconstriction because of the lower osmotic pressure within droplets deposited within the airways than that of cells lining the airways (hypoosmolarity). Water consequently will pass from deposited droplets into adjacent cells, disrupting cell function (Folinsbee, 1989).

Table 7.1 Summary of results of human volunteer experiments with sulphuric acid

Study	Concentration H ₂ SO ₄ µgm ⁻³	Duration (minutes)	Effects
Utell <i>et al</i> (1984)	1000	16	increase in bronchial responsiveness of normal volunteers to carbachol
Avol <i>et al</i> (1988)	1000	60	no effect on FEV ₁ , symptoms or responsiveness to methacholine
Utell <i>et al</i> (1983)	1000 450	16	decrements in FEV ₁ in asthmatics increased airways resistance in asthmatics
Koenig <i>et al</i> 1989	68	40	decrements in FEV ₁ in asthmatic adolescents
Spektor <i>et al</i> (1989)	100 100	60 120	decreased rates of particle clearance greater and longer lasting effects on clearance
Koenig <i>et al</i> 1993a	35	45	decrements in FEV ₁ in asthmatic adolescents
	70	45	decrements in FEV ₁ in asthmatic adolescents
	35	90	no effects on FEV ₁ in asthmatic adolescents
	70	90	no effects on FEV ₁ in asthmatic adolescents
Koenig <i>et al</i> 1993b	70	40	no effect in older subjects including asthmatics
Leduc <i>et al</i> (1996)	500	60	no effects on pulmonary function or bronchial responsiveness

There are two main mechanisms by which the airways are protected from the effects of inhaled acids. Firstly, much of the acidity is neutralised by the presence of ammonia (NH₃) within the airways and secondly, acidity is also buffered by interaction with airways mucus (Folinsbee, 1989). Experimental data suggests that normal levels of oral NH₃ should generally be sufficient to neutralise ambient concentrations of H₂SO₄ (Larson *et al*, 1982, Utell *et al*, 1989). The quantity of H⁺ required to exceed the buffering capacity of airways mucous is thought to be more than 10 times greater than the quantity that would be deposited following 30 minutes inhalation of 100µgm⁻³ H₂SO₄ (Holma, 1989). More recently, Culp *et al* (1995) found that inhalation of 1000 µgm⁻³ sulphuric acid had no effect on the glycoproteins and proteins present in mucus lavaged from human volunteers. COMEAP (1995) suggested that the neutralising capacity of the airways only allows acid to penetrate to the small airways at relatively high levels of exposure. High dose experiments therefore tend to be associated with effects on the smaller airways as indicated by changes in MMEF (maximal mid expiratory flow). At lower doses, the only lung function parameter that is associated with acid exposure is S_{Gaw} (airways conductance) suggesting effects are confined to the larger airways.

Differences in the buffering capacity of the airways under different experimental protocols may explain some of the disparity between the response of human volunteers to sulphuric acid (COMEAP, 1995). Only some investigations have specifically attempted to remove oral NH₃ prior to the exposure of volunteers to H₂SO₄. Where experimental protocols have been designed to minimise oral NH₃, there is little evidence that exposure to sulphuric acid concentrations of 500µgm⁻³ is associated with decrements in the lung function of normal subjects (Wyzga and Folinsbee, 1995). It has been suggested that asthmatic adolescents are more susceptible to the effects of acid aerosols in exposure experiments than adults because of the limited buffering capacity of their airways (Folinsbee, 1989).

A further aspect of experimentation with acid aerosols that may contribute to the contradictory results of different studies is particle size. Particle size affects both the efficiency and location of acid deposition within the airways. Large fog particles such as might arise at the highest concentrations tend to deposit in large airways giving rise to upper respiratory symptoms without affecting lung function (Folinsbee, 1989). Fine *et al* (1987), for example, found a smaller effect associated with exposure to 10000 μgm^{-3} of H_2SO_4 than Utell *et al* (1983) found with 1000 μgm^{-3} H_2SO_4 . The particles in the fog created by Fine *et al*, however, had a mass median aerodynamic diameter of $6\mu\text{m}$ and were largely not respirable whereas the particles created by Utell *et al* were only $1\mu\text{m}$ and highly respirable. Whereas the larger droplets used in the fog experiment would be typical of naturally occurring fogs, the smaller more respirable particles used by Utell *et al* (1983) would be more typical of those present in fog-free air. The sodium sulphate particles in emissions from glass furnaces and any associated sulphuric acid-salt particles would generally be in the $1\mu\text{m}$ size range with larger particles only forming in foggy weather.

Overall there is a stronger association between sulphuric acid and adverse effects in human volunteer experiments than with other sulphate species. The increment in aerosol acidity associated with emissions from glass furnaces is, however, likely to be much smaller than that associated with measurable effects in human volunteer experiments.

7.5 COMBINED EXPOSURES TO ACID AEROSOLS AND OXIDANT GASES

Given the lack of evidence of a dose response effect with sulphuric acid alone, MAAPE (1992) suggested that the increased bronchial responsiveness induced by sulphuric acid may potentiate the effect of other pollutants, constrictor stimuli or antigens. High levels of acidity can be generated in combination with high ozone concentrations during summertime photochemical pollution events. Frampton *et al* (1995a and b) and Utell *et al* (1994) found that the pre-exposure of subjects to $100\mu\text{gm}^{-3}$ sulphuric acid is associated with decrements in lung function on exposure to 80ppb ozone the next day in asthmatic but not normal subjects. Linn *et al* (1993, 1994) found that combined 5 hour exposures to sulphuric acid ($100\mu\text{gm}^{-3}$) and O_3 (120ppb) appeared to have non- or marginally significant synergistic effects on lung function in both normal and asthmatic volunteers. The exposures were associated with a greater increase in airways resistance (S_{raw}) and a greater decrement in FEV_1 in asthmatic volunteers than in subjects without asthma. Subsequently, Linn *et al* (1995) exposed asthmatic volunteers aged 11-18 to respirable acid aerosol in combination with 300ppb NO_2 and 200ppb ozone. The H^+ concentration ($2500\text{nmolH}^+\text{m}^{-3}$) was considerably higher than is typical of photochemical haze and exposures were not significantly associated with lung function changes in the group as a whole. A few subjects, however, showed decrements in lung function that could be indicative of a more susceptible subgroup. Linn *et al* (1997) exposed children aged 9 to 12 to a simulated acid summer haze with concentrations of ozone of 100ppb, SO_2 100ppb and sulphuric acid 60-140 μgm^{-3} for 4 hours with intermittent exercise. They found a nonsignificant effect in terms of decreased lung function or increased symptoms. There was a positive association between symptoms and acid dose in subjects with asthma or allergy, but the effect was much smaller than that found in epidemiological studies.

Overall it appears that exposure to acid aerosols in combination with ozone may be associated with a larger decrement in lung function than would arise solely through exposure to ozone, but the effect is largely confined to susceptible individuals. The experimental levels of acidity have been much greater than typical of those in photochemical smogs, but this may be counterbalanced by the relatively short duration of exposure. Concentrations of ozone in the UK are generally much lower than those at which synergistic effects have been found with sulphuric acid.

7.6 ACID-PARTICLE INTERACTIONS

There has been some interest in the role of surface adsorbed acidity in causing adverse effects associated with inhaled particles. A study of the effects of sulphuric acid adsorbed on inhaled carbon black particles in 30 human volunteers, however, showed no significant association of effects on lung function (FEV₁, FVC) and concentrations equivalent to 100 µgm⁻³ of H₂SO₄ (Anderson *et al*, 1992). The effects on airways resistance (S_{raw}) were no greater than for separate exposure to 200µgm⁻³ carbon black or 100µgm⁻³ acid.

7.7 OTHER SOLUBLE PARTICLES

7.7.1 Sulphite

Utell *et al* (1984) reported that exposure to 1000µgm⁻³ of sodium sulphite was associated with a significant increase in methacholine responsiveness in terms of sGaw (airways conductance) and to a lesser extent MMEF. MAAPE (1992) in reviewing the potential effects of secondary particles found that sulphite, bisulphite and metasilphite caused a greater degree of bronchial constriction in asthmatics than SO₂ or acidity. They concluded that this response was related to the production of SO₂, which causes acute effects in asthmatics (MAAPE, 1992).

7.7.2 Sodium chloride

Like sodium sulphate, sodium chloride is a highly soluble neutral salt. It has been used as a control particle in some human volunteer experiments. Koenig *et al* (1980, 1981), Morgan *et al* (1977) and Kleinman and Bailey (1985) have all demonstrated that short term exposure to 1000µgm⁻³ of sodium chloride has no significant effect on lung function in either healthy adults or asthmatic adolescents. There also found no association with respiratory symptoms. Interactions with other pollutants, however, may be important. Koenig *et al* (1980, 1981) showed that the combination of exposure to sodium chloride and 1ppm sulphur dioxide (about 100 times current ambient concentrations) is associated with decrements in lung function and wheezing in asthmatic adolescents. Kleinman and Bailey (1985) showed that combined exposures to sodium chloride and nitrogen dioxide or irritant particles (zinc ammonium sulphate) were associated with increased respiratory symptoms, although the effects were not significant.

7.7.3 Sodium nitrate

Exposure of mild asthmatics to sodium nitrate at 1000µgm⁻³ was not associated with symptoms or any change in lung function (Utell *et al*, 1979).

7.8 CONCLUSIONS

The results of human volunteer experiments with sulphate species (principally ammonium sulphate) suggests that acidity is more likely to be of relevance in the development of adverse effects than sulphate. This suggests that no adverse respiratory effects would be expected to be associated with exposures to sodium sulphate particles at the predicted ambient levels. There is a possibility, however, that sodium sulphate emissions from glass furnaces could provide nuclei for the formation of acid aerosol, particularly given the presence of sulphur dioxide in the waste gas stream. There is limited evidence from human volunteer experiments to suggest an association between exposure to acid aerosol and health effects, with asthmatic adolescents being the most susceptible members of the population. The size of inhaled acid particles would have a critical effect on the effective dose. Under most conditions, particles might be approximately in the 1µm size range and have a similar biological activity to those

used in some experimental systems. Under conditions of relatively high humidity, however, acid fogs may form with coarser particle sizes and consequently much lower rates of deposition in the airways and less effect on lung function.

The hygroscopic growth of soluble particles such as sodium sulphate or sulphuric acid within the human respiratory tract is likely to lead to increased deposition within the upper airways and reduced penetration to the deep lung relative to insoluble particles. This may be significant to understanding specific respiratory effects that may be associated with soluble particles as opposed to PM_{10} more generally.

8. SODIUM SULPHATE TOXICITY: EPIDEMIOLOGY

8.1 INTRODUCTION

There is only one specific study of the health effects of environmental exposure to sodium sulphate and one workplace study. There is, however, a considerable literature regarding exposure to secondary sulphate particulate including acid aerosol in the general environment. Interest in the potential effects of aerosol acidity was particularly marked during the 1980s. Renewed interest in soluble secondary particles has arisen following the review by Lippmann and Thurston (1996) that concluded that sulphate has generally proved a better indicator of adverse health effects than other measures of particle concentrations. Sulphate is present in body fluids at relatively high concentrations and it therefore seems unlikely that small increments in sulphate uptake by the airways would cause adverse effects. Lippmann and Thurston (1996) suggested that the apparent importance of sulphate may have arisen because it is a surrogate for both PM_{2.5} and acidity. There is generally a good correlation between environmental concentrations of secondary sulphate and aerosol acidity, particularly in eastern North America where most of the relevant studies have been undertaken (Oskaynak *et al*, 1996; Spengler *et al*, 1996). This has made it difficult to determine an independent effect for sulphate as opposed to acidity in epidemiological studies of the effects of air quality. This chapter describes the outcome of epidemiological studies of the specific effects of sodium sulphate and reviews evidence for a more general association between exposure to soluble particles and health.

8.2 WORKPLACE EXPOSURE TO SODIUM SULPHATE

There is no specific workplace limit for sodium sulphate either in the UK or elsewhere. In the UK workplace concentrations of respirable dusts for which no specific limit has been set should be controlled to 4mgm⁻³.

Kelada and Euinton (1978) examined 119 workers exposed to sodium sulphate dust during mining operations in Saskatchewan. The health end points investigated included a range of subjective symptoms and also measured properties such as lung function, serum sulphate, calcium and electrolytes. There was no difference in health status between workers with less than or more than ten years exposure to the dust. The only symptom that appeared to be associated with the dust was nasal irritation and associated runny nose. There was no evidence, however, of nasal septum perforation which has been reported to occur as a result of exposure to other hygroscopic dusts including sodium chloride, potassium chloride and sodium carbonate (Kelada and Euinton, 1978). Measured concentrations of sodium sulphate ranged from less than 5mgm⁻³ to 150mgm⁻³. No specific exposure information was presented in the study, but exposure concentrations are likely to have been several orders of magnitude greater than those associated with emissions from glass furnaces.

There has been little investigation of the effects of workplace exposure to other near neutral sodium or sulphate salts. Tarchi *et al* (1994) in a study of rock salt miners found evidence of an association between increased cancer risks and exposure to chrysotile (white asbestos) but no specific effects associated with exposure to rock salt per se. Burilkov and Michailova (1990) in a study of gypsum workers found no evidence that exposure to concentrations of up to 30 mgm⁻³ of dust containing gypsum was associated with pneumoconiosis but some evidence of an association with bronchitis. The dust, however, also contained carbonate, clay, feldspar and quartz.

Overall, the limited workplace information available suggests that it is highly unlikely that exposure to sodium sulphate at the concentrations likely to arise as a result of emissions from glassworks will have a measurable effect on human health.

8.3 ENVIRONMENTAL EXPOSURE TO SODIUM SULPHATE

Environmental exposure to airborne sodium sulphate has occurred where shallow saline lakes have dried out to leave surface salt deposits that are subject to wind erosion.

Micklin (1988) reported that desiccation of the Aral Sea in the former Soviet Union was associated with throat cancer and respiratory and eye problems in local residents. It is possible that these symptoms are associated with windblown salt and dust from the lake bed. Windblown salt has had a demonstrable adverse effect on vegetation around the Aral Sea whereas there is no evidence of phytotoxicity associated with sodium sulphate emissions from glassworks (Chapter 9). Micklin (1998) provides no information about airborne concentrations of sodium sulphate in areas down wind of the Aral sea, but the phytotoxic effects, suggest concentrations are likely to have been vastly greater than around glassworks. The adverse health effects observed in local communities may not have been simply associated with airborne salts but could also have been associated with the reduced water quality due to the reduced volume of the lake and lowering of the water table.

Gomez *et al* (1992) investigated the effects of salt particles blown off a desiccated lake bed in Saskatchewan on the health of local communities living at distances of 5 to 7km from the lake margin. There is no indication of the airborne concentrations of sodium sulphate to which these communities were exposed. Levels of sodium sulphate deposition were, however, sufficient to substantially affect soil chemistry suggesting that concentrations were much greater than would be associated with emissions of glassworks (Chapter 9). The prevalence of current and chronic symptoms was greater in a sample of individuals from the affected communities than in individuals from a comparable community not subject to the windblown salt. The prevalence of cough and wheeze were more than double, and of nasal and eye irritation more than three times higher, in affected communities than in the control community. Lung function parameters were, however, comparable in both groups, although the number of participants limited the power of the study to detect small effects on lung function (<10%). The authors postulated that the absence of an effect on lung function was partly due to the effects of recent rain in reducing fine particle concentrations. The observed symptoms are consistent with deposition of salts within the upper respiratory tract.

Overall, there is evidence that environmental exposure to high concentrations of airborne sodium sulphate particles may be associated with upper respiratory symptoms, although the relative importance of sodium sulphate as opposed to other components of windblown dust is hard to discern. The concentrations at which such effects have been observed are several orders of magnitude greater than those likely to arise as a result of emissions from glassworks.

8.4 EPIDEMIOLOGICAL INVESTIGATIONS OF THE EFFECTS OF SECONDARY SOLUBLE PARTICLES

8.4.1 Ambient air quality in relevant studies

Some of the difficulties in interpreting the results of epidemiological investigations of air pollution arise from the difficulty in separating the effects of different pollutants and understanding the interactions between different pollutants. In addition, there are uncertainties about the extrapolation of dose-response relationships to concentrations of air pollution that are substantially different from those prevalent in the original study. Most investigations of

the potential health effects associated with soluble particles have been undertaken in North America. Ambient concentrations of soluble particles in both Europe and North America are very small (Section 4.4.2) but there are differences in air quality between Europe and North America and also between different epidemiological studies. Air quality data for the epidemiological studies reviewed in the remainder of this chapter are summarised in Table 8.1. With the exception of the London smog studies, levels of soluble particle pollution in most studies appear to have been fairly similar to those prevalent in the UK whereas overall levels of particle pollution in some studies have been slightly higher. The main other difference in air quality between studies is with respect to ozone (Table 8.1). Levels of ozone in some North American studies have been considerably higher than would be typical of the UK. Exposure to ozone is associated with a range of adverse respiratory effects and experimental evidence also suggests that exposure to ozone may potentiate the response to other pollutants (MAPPE, 1991; Kinney *et al*, 1996).

Table 8.1 Pollutant concentrations in the reviewed studies of the association between health indicators and short term exposure to elevated levels of aerosol acidity or other secondary particle concentrations. Unless otherwise indicated data shown are the mean (standard deviation) of the 24 hour daily average

Study	PM ₁₀ µgm ⁻³	PM _{2.5} µgm ⁻³	SO ₄ µgm ⁻³	NO ₃ µgm ⁻³	H ⁺ nmolm ⁻³	O ₃ ppb
UK range of annual mean	19-30		2-8		6-14	9-29
UK range of maximum	21-281*					51-121**
London smog 1962-74	100***				130	
The Netherlands						
Hoek and Brunekreef 1994	44(9)		6 (7)	7 (3)		
Hoek and Brunekreef 1995	42(21)		6 (7)	7 (7)		58 (20) **
North America						
Ostro <i>et al</i> 1991		22.0 (20.4)	2.0 (2.5)	6.8 (17.6)	10 (9.5)	
Thurston <i>et al</i> 1997****			7.0 (1.8)		53.9 (15.2)	84 (10.2)**
Delfino <i>et al</i> 1994	29.5 (12.0)		4.2 (3.7)			59.7 (31.1) 72.1 (34.5)**
Delfino <i>et al</i> 1997	30.1 (17.3)	18.5 (14.0)	5.2 (7.9)		11.3 (19.9)	
Burnett <i>et al</i> 1997			5.7 (7.4)		5.0 (9.4)	
Burnett <i>et al</i> 1999	30.2 (13.6)	18.0 (8.5)				19.5 (14)
Schwartz <i>et al</i> 1994	30 (10)	18 (9)	7.5 (4.5)		18.1 (15)	36.9 (15)
Austria						
Studnicka <i>et al</i> 1995*****	10.3 (4.4)		8.4. (3.6)		32.2 (16.9)	56.4 (8.2)

* maximum daily

** mean maximum hourly

***black smoke

****afternoon

***** pollution data for panel 1 for which the strongest association with health effects was found

8.4.2 Acute Mortality

A number of studies have shown associations between sulphate or acidity and daily mortality. Acidity, for example, may have played an important role in the association between daily mortality and air quality observed for London smogs during the 1950s and 1960s (Ito and Thurston 1989; Ito *et al* 1993; Lippmann and Thurston 1996; Lippmann and Ito 1995). These studies showed a stronger association of acute mortality with acidity than with SO₂ or black smoke during the winter (November to February). During the rest of the year the strongest association was with SO₂. Mean concentrations of SO₂, black smoke and acidity were vastly greater than now found anywhere in the UK including areas impacted by glassworks.

In contrast to the London studies, Pope *et al* (1992) report an association between daily mortality and respirable particulate pollution measured as PM₁₀ in a study of Utah Valley in the absence of substantial levels of SO₂ or aerosol acidity. This suggests an independent association between mortality and PM₁₀ and a limited role for aerosol acidity. Similarly, Dockery *et al* (1992) in a study of three US cities found the strength of association with daily mortality was weaker for sulphate and acidity than for PM₁₀ and PM_{2.5}. The correlation between sulphate and H⁺ and PM₁₀ (r=0.52, 0.76) did however, weaken the power of the study to find independent effects for each of the three measures of particulate. Schwartz *et al* (1996)

in a study of six US cities found that concentrations of PM₁₀, PM_{2.5} and sulphate were significantly associated with daily mortality while no associations were found with coarse mass (TSP-PM₁₀) or aerosol acidity. The potential importance of the fine fraction of PM₁₀ was also demonstrated in a recent study by Schwartz *et al* (1999) of the effects of dust storms in Washington State. They found that there was no association between daily mortality and the coarser component of PM₁₀ that dominates airborne particulate during dust storms.

Overall, although there is a well established association between daily mortality and ambient particle concentrations, there is no consistent evidence that soluble particle concentrations or acidity are more strongly associated with daily mortality than other components of PM₁₀ or PM_{2.5}. There is evidence that the finest fractions of PM₁₀ are more strongly associated with mortality than coarser material in the PM₁₀ size range. Both the initial size of inhaled soluble particles and the extent to which their size is modified by hygroscopic growth within the respiratory tract may have an important influence on acute effects, including mortality.

8.4.3 Short term changes in lung function

Lipfert (1994) in reviewing a number of studies found considerable evidence of an association between air pollution and short term decrements in lung function. The most extensive investigations of possible associations between lung function and soluble particles have been performed for children attending summer camps in the US exposed to the effects of photochemical smog. The findings of these and some other relevant studies are shown in Table 8.2. There are a number of difficulties in the interpretation of lung function studies including the possible confounding effects of passive smoking and exposure to allergens such as pollen and fungal spores. Aerosol acidity may potentiate the effects of ozone in causing decrements in lung function associated with summertime haze. Asthmatic children appear to be more susceptible to the effects of acidity and/ or sulphate than other children.

Table 8.2 Change in lung function (ml or mls⁻¹) in relation to incremental increases of a range of pollutants

Study	Endpoint	H ⁺ nmolm ⁻³	SO ₄ µgm ⁻³	PM ₁₀ µgm ⁻³	other comments
Children (Photochemical smog)					
Raizenne <i>et al</i> 1989	PEF FEV ₁	none	2 1		effects only seen in subjects identified as bronchially responsive
Studnicka <i>et al</i> 1995	PEF FEV ₁	none 0.4	none 0.8	1.33	
Neas <i>et al</i> 1995	PEF (all) PEF(asthmatics)	0.14 0.18		1.0 1.3	
Neas <i>et al</i> 1996	PEF	0.06		0.003	
Neas <i>et al</i> 1999	PEF		5.1		
Thurston <i>et al</i> 1997	PEF(asthmatics)	1.3	6		
Children (Wintertime pollution)					
Hoek and Brunekreef 1995	PEF FEV ₁		5 0.97		
Adults (Photochemical smog)					
Korrick <i>et al</i> 1998	FEV ₁ FVC PEFR	0.03 0.03 0.1		0.6* 0.6* 1.4*	
Naether <i>et al</i> 1999	PEF	0.3	0.2	1.2*	

*effect with PM_{2.5} rather than PM₁₀

The results of the studies shown in Table 8.2 suggest that mole for mole, concentrations of acidity have a greater influence on lung function than sulphate. An increase of 1µgm⁻³ in concentrations of sulphate, PM_{2.5} or PM₁₀ is associated with a less than 2mls⁻¹ change in PEF or 2ml change in FEV₁. An increase of 1nmol⁻³ in H⁺ (equivalent to 0.05µgm⁻³ H₂SO₄) is associated with an approximate 1mls⁻¹ change in PEF and less than 0.5ml change in FEV₁. It is not clear that sulphate particles have an independent effect on lung function from that associated more generally with particle concentrations or that sulphate is any less hazardous than other particle types. In terms of sodium sulphate emissions from glassworks, the maximum increment in local concentrations of PM₁₀ that is likely to occur under conditions of extremely poor dispersion might be associated with a less than 50mls⁻¹ change in PEF or 50ml change in FEV₁. This represents a short term decrement in lung function of less than 1% and would have no perceptible long term effect on health. The formation of acid aerosol from sulphur dioxide emissions under conditions of particularly poor dispersion might have a slightly greater, but still small, short term effect on lung function.

8.4.4 Acute respiratory symptoms

A number of studies have shown that exposure to PM₁₀ is associated with an exacerbation of respiratory symptoms, particularly in asthmatics and others with pre-existing respiratory illness (COMEAP, 1998). Few investigations have been made of the specific role of soluble sulphate particles. Ostro (1990) showed that the number of restricted activity days in US citizens due to respiratory illness was more strongly associated with ambient sulphate concentrations than with other surrogate measures of particle concentrations. Ostro *et al*

(1991) studied a group of asthmatics in Denver, Colorado during the winter months. They found that a $10\mu\text{gm}^{-3}$ increase in sulphate was associated with a 0.77% ($\pm 0.42\%$) increase in the incidence of cough symptoms. A 10nmolm^{-3} increase in H^+ was associated with a 2.0% ($\pm 0.2\%$) increase in the incidence of breathlessness. Their results suggested an independent role for acidity but the specific importance of sulphate was less clear. A more recent study of wintertime pollution in the Netherlands found no significant association between exposure to PM_{10} , sulphate, sulphite, or nitrous acid and respiratory symptoms in children (Hoek and Brunekreef, 1994). Both PM_{10} and sulphate showed nonsignificant associations with cough. Nitrate and sulphate both showed nonsignificant associations with lower respiratory symptoms. Peters *et al* (1997b) reported an association between 5 day mean concentrations of sulphate and symptoms in children with asthma in the Czech Republic.

Studies of summertime photochemical pollution in both American and Dutch children have similarly shown inconsistent relationships between respiratory symptoms and soluble particles (Table 8.3).

Table 8.3 Increase in incidence in respiratory symptoms in children associated with air pollution (on previous day) during summertime photochemical episodes

Study	Increment in pollution	Cough	Lower respiratory symptoms
Schwartz <i>et al</i> , 1994	$30\mu\text{gm}^{-3}$ PM_{10}	28%	53%
	$20\mu\text{gm}^{-3}$ $\text{PM}_{2.5}$	19%	44%
	$5\mu\text{gm}^{-3}$ $\text{PM}_{2.5}$ sulphur	23%*	82%
	1km^{-1} Haze	21%	36%
	25nmolm^{-3} Acidity H^+	6%*	5%*
Neas <i>et al</i> 1996	125nmolm^{-3} H^+	35%	
	$20\mu\text{gm}^{-3}$ PM_{10}	37%	
Hoek and Brunekreef 1994	PM_{10}	none	none
	$10\mu\text{gm}^{-3}$ SO_4	none	6%*
	$10\mu\text{gm}^{-3}$ NO_3	none	0.8%*
Thurston <i>et al</i> (1997)	sulphate and acidity	increased use of medication in asthmatic children	

*association not significant at the 5% level

Overall, the increase in prevalence of cough associated with sulphate, acidity and PM_{10} appears to be roughly in proportion to the relative concentrations of these pollutants in ambient air (Table 8.4). There is insufficient information from these studies to determine whether sulphate is more or less hazardous than other components of PM_{10} .

Table 8.4 Approximate increases in the prevalence of acute respiratory symptoms associated with secondary particles as reported in the studies described above

Increment in pollutant	Increase in incidence of cough
$1\mu\text{gm}^{-3}$ PM_{10}	1%
$1\mu\text{gm}^{-3}$ SO_4	0.8-5%
1nmolm^{-3} H^+	0.2-1.5%

8.4.5 Hospital Admissions

A number of studies have examined the association between emergency hospital admissions and air quality. Most have concluded that there is a strong association between particulate concentrations and admission for respiratory and cardiac causes (Table 8.5). The evidence for an association between concentrations of NO₂ and SO₂ and hospital admissions is generally weaker. Relatively few studies have investigated the possible association between soluble particle concentrations and hospital admissions. Where the role of sulphate has been investigated, sulphate has generally been used as a surrogate for fine particulate generally and few of the studies are directly informative about the role of soluble particles versus insoluble particles.

Table 8.5 Approximate percentage increase in risk of hospital admission associated with unit increases in air pollutant concentrations (standard error) in some example studies

increment	O ₃ ppb	H ⁺ nmol	SO ₄ µgm ⁻³	TSP µgm ⁻³	PM ₁₀ µgm ⁻³	PM _{2.5} µgm ⁻³
North American studies						
Delfino <i>et al</i> 1994					0.2 (0.05-0.4)*	
Delfino <i>et al</i> 1997	0.194** (0.058)	0.194*** (0.74)	0.047** (0.021)		0.197*** (0.073)	0.257** (0.021)
Burnett <i>et al</i> 1995						
respiratory			0.3			
cardiac			0.2			
Burnett <i>et al</i> 1997						
respiratory	0.6	4.6	0.07	0.21	0.4	
cardiac	0.6	4.6	0.04	0.23	0.3	
Stieb <i>et al</i> 1996	0.21 (0.13)		-0.030 (0.022)	0.016 (0.006)		
European studies						
Schwartz and Morris (1995)					0.099	
Polonieki <i>et al</i> (1997)					0.070	
Wordley <i>et al</i> (1997)					0.043	

*95% confidence limit
 ** adults over 64 years
 *** children under 2 years

It is difficult to compare the findings of different studies shown in Table 8.3 because of differences in local medical practice with respect to the availability of different types of treatment service. The Burnett *et al* (1997) and Delfino *et al* (1997) data suggest that the increase in hospital admissions associated with sulphate relative to that associated with PM₁₀ is roughly in proportion to, or possibly smaller than, the proportion of sulphate in PM₁₀. The results of these studies also suggest that the increase in hospital admissions associated with aerosol acidity relative to that associated with PM₁₀ is slightly greater than the proportion of acid aerosol in PM₁₀. In general, the association between aerosol acidity and risk of admission for respiratory illness is most marked in North American studies at very high levels of acidity (>100nmolm⁻³ H⁺) during photochemical smog episodes (Thurston *et al*, 1992,

1994). At lower acid aerosol concentrations, ozone appears to be more strongly associated with adverse effects. The data suggest that aerosol acidity may potentiate the adverse effects associated with exposure to ozone. Overall the risks of hospital admission associated with the predicted increments in sodium sulphate concentrations associated with glassworks would be expected to be extremely small. Under conditions of particularly poor dispersion, it is possible that acid aerosol formed from sulphur dioxide emissions would lead to a small increased risk of hospital admission for local communities.

8.4.6 Chronic mortality

There is consistent evidence from a number of studies of an association between airborne particles and a small loss of life expectancy (Pope *et al*, 1995a). Pope and Dockery (1996) concluded that there was an excess risk of mortality of between about 3% and 9% associated with an increment of $10\mu\text{gm}^{-3}$ PM_{10} compared with an estimated risk of 0.8% per μgm^{-3} PM_{10} for environmental tobacco smoke (ETS; Lipfert, 1994). The highest excess risks have been found in prospective cohort studies where it has been possible to control for confounding factors such as smoking at the individual level (Table 8.6). There have not been specific studies of the possible association between sulphate or aerosol acidity and mortality, but Pope *et al* (1995b) used sulphate as an index of particle concentrations (Table 8.6). The apparent risk of premature mortality (all cause) associated with an increment of $19.9\mu\text{gm}^{-3}$ sulphate was similar to that associated with an increment of $24.5\mu\text{gm}^{-3}$ fine particles, but there was a greater association of sulphate with lung cancer. This might reflect the importance of species such as PAHs in combustion-generated particulate rather than being specifically related to sulphate.

Table 8.6 Estimated risks of chronic mortality in US cohort studies

Study	Increment in pollutant	Mortality	Odds ratio (95% ci)
Abbey <i>et al</i> 1999	$\text{PM}_{10}>100\mu\text{gm}^{-3}$ on 43 days/year	all (men)	1.12 (1.01-1.24)
		all (woman)	0.94 (0.86-1.06)
		cardiopulmonary (men)	1.09 (0.95-1.24)
		cardiopulmonary (woman)	0.90 (0.80-1.01)
		non-malignant respiratory disease (men)	1.28 (1.03-1.57)
		non-malignant respiratory (women)	1.10 (0.91-1.33)
		lung cancer (men)	2.38 (1.42-3.97)
		lung cancer (woman)	1.08 (0.55-2.13)
Dockery <i>et al</i> 1993	46 μgm^{-3} TSP 40 μgm^{-3} PM_{15} 25 μgm^{-3} $\text{PM}_{2.5}$ 11 μgm^{-3} sulphate	all	1.26 (1.08-1.47)
Pope et al 1995b	19.9 μgm^{-3} sulphate	all	1.15 (1.09-1.22)
		cardiopulmonary	1.26 (1.16-1.37)
		lung cancer	1.36 (1.11-1.66)
	24.5 μgm^{-3} fine particles	all	1.17 (1.09-1.28)
		cardiopulmonary	1.31 (1.17-1.46)
		lung cancer	1.03 (0.80-1.33)

8.4.7 Long term loss of lung function

There is relatively little information about the specific relationship between long term exposure to soluble particles and lung function. A number of North American studies have examined lung function in areas affected by photochemical smogs. Stern *et al* (1994) found a small difference in lung function in children in Ontario relative to those in Saskatchewan that they attributed to higher levels of secondary particles. Raizenne *et al* (1996) in a study of 24 North American cities found a significant decline in lung function associated with particle acidity (3.4%/52nmolm⁻³) and weaker associations with sulphate or other measures of particle concentrations. Abbey *et al* (1998a) in their study of lung function in adult nonsmokers (Seventh Day Adventists) found sulphate to be the best predictor of effects and confirmed an independent role of sulphate as opposed to PM₁₀. The sulphate data had, however, been collected over a much longer time period, so that an alternative explanation could be that long term exposure to particulate pollution has a greater effect on lung function than medium term exposure. Abbey *et al* (1998a) did not examine the role of aerosol acidity.

Jedryschowski and Kryanowski (1989) in a cross sectional study of adults in Warsaw found an association between secondary sulphur pollutants and FEV₁ that they interpreted as an association with acid aerosol rather than with sulphur dioxide or particles. Lipfert (1994) in reworking data from a French study of lung function (cited as PAARC, 1982) found an association between lung function (FEV₁) and acid aerosol in both children and adults. The effects appear to have been stronger in children. The data suggest that HCl and HNO₃ were important in addition to H₂SO₄ which was the dominant acid species. Peters *et al* (1999a) also found a significant relationship between lung function in Californian school girls (but not boys) and chronic exposure to acid vapour (HCl and HNO₃). The effects of acid were considerably greater per unit mass than PM₁₀.

Overall there would appear to be more evidence to suggest a possible association between long term exposure to aerosol acidity and loss of lung function than with sulphate more generally.

8.4.8 Chronic respiratory symptoms

There have been few studies of the specific effects of soluble particles on respiratory symptoms. Dohan *et al* (1962) showed that the number of cases of respiratory illness in female factory workers in five US cities was strongly correlated with sulphate concentrations. Dockery *et al* (1989) found a nonsignificant association between respiratory symptoms in children in six US cities and concentrations of sulphate. Speizer (1989) showed preliminary data from four of the cities suggesting an association between the prevalence of bronchitis and long term exposure to acid aerosols. Subsequently Dockery *et al* (1996) found that children living in 24 cities in the eastern US with high levels of particle strong acidity were more likely to have had bronchitis during the last 12 months than those in less polluted areas. They were unable to show that sulphate, acidity and particles had independent effects. Asthma, persistent wheeze, chronic cough and chronic phlegm were not significantly associated with acid aerosols or other measures of particulate pollution.

In a Californian study of nonsmokers, Abbey *et al* (1998b) found no association between symptoms and sulphate. In a further Californian study, Peters *et al* (1999b) found an association between wheeze in boys and acid vapour (OR = 1.45; 95%CI 1.14-1.83 for a range of about 4nmolm⁻³ as HCl and HNO₃). In a further study of children in the same area of south California, McConnell *et al* (1999) found statistically significant associations between PM₁₀ and bronchitis and concentrations of PM₁₀, PM_{2.5}, NO₂ and acid (H⁺) and phlegm in

asthmatic children. Respiratory symptoms in children without a history of wheezing and asthma were not associated with air quality.

Overall, there appears to be some evidence of an association between long term exposure to acid aerosols and respiratory symptoms, particularly in people with asthma. An increment in aerosol acidity of about 10nmol H^+ is associated with an increase of up to 10% in the prevalence of bronchitis in children. There is, however, more consistent evidence of an association between concentrations of PM_{10} and respiratory symptoms. There is no consistent evidence for an independent role for sulphate as opposed to other types of particle.

8.4.9 Identification of an independent role for sulphate particulate

Overall it is difficult to unravel the relative importance of soluble particles relative to less soluble components of PM_{10} as a possible cause of adverse health effects. There is limited evidence that exposure to acid aerosols is associated with acute health effects in some susceptible individuals. There is also limited evidence that long term exposure to acid aerosol is associated with a small adverse effect on respiratory health. In most studies where sulphate concentrations are quoted, however, they have been used as a surrogate for fine particulate. It is therefore difficult to determine the relative importance of particle size and particle composition. In the UK concentrations of sulphate are generally less than 20% of total PM_{10} whereas US investigators have used a conversion factor of $\text{PM}_{10} = \text{sulphate} \times 4$ (Dockery and Pope, 1996) to relate the results of different studies. Given these factors, the apparent relationships between adverse health effects and sulphate concentrations could be largely explicable in terms of an association between health and particles generally rather than specifically with sulphate (Table 8.7). The associations between sulphate and health effects seen in epidemiological studies could largely reflect the generally close correlation between sulphate concentrations and fine particulate concentrations. Even where apparent effects seem to be more closely associated with sulphate than with other measures of fine particulate, it is possible that sulphate concentrations could be a proxy for aerosol acidity or some other relevant pollutant. The proportion of secondary particles present in urban air is likely to be very different in Europe, Latin America and the US, yet studies of acute mortality show a fairly consistent relationship between concentrations of PM_{10} and excess mortality (Brauer *et al*, 1995). This tends to suggest that aerosol composition is of less importance than particle size.

Table 8.7 Comparison of effects associated with airborne sulphate concentrations with those attributed to PM₁₀

Effect	Study/review	Approximate change per 2.5µgm ⁻³ SO ₄	Approximate change per 10µgm ⁻³ PM ₁₀
chronic mortality	Dockery <i>et al</i> 1993	0.25-0.5 years	
years shortening of life	COMEAP 1998		0.4 years
acute hospital admission respiratory	Burnett <i>et al</i> (1995) studies cited by COMEAP	0.7%	0.4-2.0%
cardiac	Burnett <i>et al</i> (1995) studies cited by COMEAP	0.4%	0.4-1.0%
morbidity			
bronchitis (children)	Dockery <i>et al</i> (1996)	24%	
emphysema, chronic bronchitis or cough	Pope and Dockery (1996)		10-25%
lung function acute			
adults	Naether <i>et al</i> 1999 PEF	0.025	
children	Pope <i>et al</i> (1991) PEF Hoek <i>et al</i> (1996) PEF		0.2-0.4% 0.1%
lung function (chronic)			
adults	Abbey <i>et al</i> (1998a) Pope and Dockery (1996)	2.4%	2% <2%
children	Stern <i>et al</i> (1994)	0.7%	

8.5 OVERALL ASSESSMENT OF THE EPIDEMIOLOGICAL EVIDENCE OF A POTENTIAL ASSOCIATION BETWEEN SODIUM SULPHATE AND HEALTH

The limited workplace information about sodium sulphate suggests that it has a low toxicity and is not associated with serious adverse effects, even at quite high levels of exposure. There is no evidence of pneumoconiosis such as can be observed with insoluble dusts such as cement or coal, but this could be partly due to the limited investigation that has been made of exposed workers. There is some evidence for irritation of the upper respiratory tract but at concentrations that are likely to be orders of magnitude higher than those associated with emissions from glass furnaces.

The limited information about the effects of exposure to high concentrations of sodium sulphate in the environment suggests that it could be associated with upper respiratory symptoms and eye irritation. It is likely that the levels of exposure associated with these symptoms are hundreds to thousands of times higher than likely levels of exposure associated with glass furnace emissions.

There is little epidemiological evidence to suggest that neutral sulphate salts are more strongly associated with adverse health effects than other components of particulate air pollution. As with the toxicological studies, the effect seems to be more strongly associated with acidity. There is, however, no evidence to confirm that sulphate is any less harmful than other components of particulate air pollution.

The results of epidemiological studies of the potential effects of exposure to soluble sulphate phases largely formed as a result of secondary processes in the atmosphere suggest that aerosol acidity may be linked with some respiratory symptoms and loss of lung function. Effects are greatest during photochemical smog when exposures to acid aerosol are combined with

relatively high exposures to ozone. Sulphur dioxide emissions from Pilkington's St Helens works are likely to give rise to a small increment in local concentrations of aerosol acidity under unfavourable atmospheric conditions. This small increment in secondary soluble particle concentrations might give rise to a small increase (<0.5%) in the prevalence of respiratory symptoms. It is not likely to give rise to measurable effects in local communities.

Particle size and the growth of hygroscopic particles in the respiratory tract play an important role in determining particle deposition and potentially the associated health effects. There is some evidence that the finest component of PM₁₀ is more strongly associated with daily mortality than the coarse component of PM₁₀ (Schwartz *et al*, 1999). In contrast the coarse component of PM₁₀ as found in volcanic ash is associated with the exacerbation of asthmatic symptoms (Cloughery *et al*, 1997; Gordian *et al*, 1996). This suggests that the health effects associated with exposure to sulphate salts might be expected to be slightly different from those associated with particles more generally. Sodium sulphate might be expected to be more strongly associated with upper respiratory symptoms, cough and exacerbation of asthma than with daily mortality and long term effects on lung function.

Overall it seems possible that on one or two days in a year, when conditions are particularly unfavourable for the dispersion of glass furnace emissions, the increment in local concentrations of PM₁₀ due to primary sodium sulphate emissions and the formation of acid sulphate particles might lead to some exacerbation of symptoms in asthmatics and others with pre-existing respiratory illness. The predicted increment in annual mean concentrations of sodium sulphate is far too small to be expected to be associated with a measurable long-term effect on health.

9. OTHER ENVIRONMENTAL EFFECTS OF PARTICLE EMISSIONS FROM GLASS FURNACES

9.1 PHYTOTOXICITY

A Dutch study (van der Eerden, 1993) of the potential phytotoxic effects of sodium sulphate emissions from glassworks concluded that that effects on local rates rates of sodium and sulphate deposition were negligible. There is no specific phytotoxicity information for sodium sulphate, but sodium chloride in road salt and in sea spray is associated with plant damage. Sodium sulphate as a highly soluble salt might be expected to show similar phytoxic effects at high concentrations.

A Belgian study undertaken by the glass industry of the impact of glassworks on vegetation found no evidence of phytotoxicity associated with emissions (Beerkens *et al*, 1998). Glassworks contributed little to local levels of industrial pollutants including sulphur dioxide, hydrogen fluoride and heavy metals. Although concentrations of lead in vegetation were higher than the regulatory limits, the main source of lead was not the glass industry. Similarly a German study cited in the same review found no evidence of damage to the health of trees in the vicinity of a Bavarian glassworks.

Overall there is no evidence to suggest that emissions from glassworks have a measurable effect on the health of local vegetation. Although the deposition of sodium sulphate on vegetation might be expected to have an adverse effect, the levels associated with emissions from glassworks are far too small to be expected to have any identifiable effect. Both sodium and sulphate are present in ambient aerosol and rainwater and the increment in concentrations associated with emissions from glass works is too small (<<5%) to cause observable damage to plants.

10. HEALTH EFFECTS ASSOCIATED WITH GASEOUS EMISSIONS FROM GLASSWORKS

10.1 INTRODUCTION

Sulphur dioxide and NO_x are emitted in substantial quantities from glass furnaces. Both pollutants have been shown to be associated with adverse effects on health in both experimental and epidemiological studies and are regulated under the UK Government's Air Quality Strategy. It is difficult to determine the scale of any effects that can be attributed to these gaseous pollutants as their main sources are similar to those of particles. Episodes of elevated concentrations of SO₂ and of NO_x tend to coincide with high levels of particles. There is a growing belief that particles are the most important component of air pollution in terms of the reported association between air quality and health (COMEAP, 1998). The extent to which pollutants such as SO₂ and NO_x may enhance the effects of ambient particles or independently cause effects is unclear.

10.2 SULPHUR DIOXIDE

Exposure to high concentrations of sulphur dioxide relative to ambient levels is associated with respiratory irritation, particularly in individuals with asthma. Experiments with human volunteers have shown effects at concentrations equivalent to those that may arise during high pollution episodes (MAAPE, 1992).

A major collaborative European study (APHEA) found apparent relationships between SO₂ and both acute mortality and hospital admissions for respiratory symptoms in elderly patients (Katsouyanni *et al*, 1997; Spix *et al*, 1998). There is considerable evidence of an association between SO₂ and the exacerbation of acute symptoms in asthmatics (MAAPE, 1992, Sunyer *et al*, 1997) but the long term significance of such effects is unclear. There is limited evidence that prolonged exposure to elevated concentrations of SO₂ in UK urban areas prior to the introduction of smokeless zones during the 1960s was associated with some shortening of life expectancy associated with bronchitis (Lipfert, 1994). Current concentrations of SO₂ in the UK are much lower than those anticipated to cause exacerbated symptoms or long term effects in healthy adults. The predicted increments in concentrations of SO₂ associated with emissions from glassworks are unlikely to lead to exceedance of the Government's air quality objectives (Table 4.10) and would therefore not be expected to be associated with adverse health effects, even in susceptible individuals. The increment in long term mean concentrations of SO₂ due to emissions from glassworks is very small (Table 4.10) and would not be expected to be associated with an adverse effect on human health.

10.3 OXIDES OF NITROGEN

There has been considerable research into the possible association between adverse health effects and exposure to NO_x, both with respect to NO_x emissions from vehicles and with respect to NO_x emissions associated with gas cookers. Major sources of NO_x such as cooking fumes or vehicle exhaust are also major sources of particles and other pollutants such that the specific importance of NO_x is difficult to identify. Experimental studies have shown that acute exposures to NO₂ can induce bronchial hyper-reactivity to non-specific agents such as histamine, methacholine and cold air, but does not induce significant alterations in lung function (MAAPE, 1993). Experimental studies also suggest that asthmatics are more susceptible to the effects of NO₂ than normal subjects. Epidemiological studies have suggested that there may be an association between NO₂ and hospital admission for some respiratory conditions (Chronic Obstructive Pulmonary Disease and Asthma; Anderson *et al*,

1997; Sunyer *et al*, 1997) but not for respiratory causes generally (Spix *et al*, 1998). Epidemiological studies have also suggested a possible association between NO₂ and acute mortality, but the apparent effects of NO₂ may be largely explicable in terms of concurrent exposure to particles (Touloumi *et al*, 1997). Similarly, although Zemp *et al* (1999) found associations between long term exposure to NO₂ and a range of respiratory symptoms, the correlation of NO₂ concentrations with those of PM₁₀ precluded the finding of an independent effect. A number of studies have examined the association of respiratory symptoms with gas cooking and found contradictory effects. The WHO (1997) in a meta-analysis found an association with respiratory symptoms in school-age children but not younger children whereas Burr *et al* (1999) found no association between gas cooking and respiratory symptoms. It is possible, also, that effects associated with gas cooking are not actually associated with NO_x exposure (COMEAP, 1998).

Overall, it is difficult to determine the relative importance of NO_x as opposed to other components of ambient air pollution in causing adverse health effects. NO₂ might be expected to have less potential to cause effects than ozone which is a stronger oxidant and SO₂ which is more soluble and therefore potentially more likely to interact with airways epithelium. The experimental evidence suggests that there is an increased risk of exacerbation of symptoms in asthmatics and others with pre-existing respiratory illness during high pollution events involving NO_x. This suggests that during periods of exceptionally unfavourable weather for dispersion, emissions from glass furnaces may lead to some exacerbation of respiratory symptoms in the local population. The epidemiological evidence linking long term exposure to NO_x to adverse health effects is equivocal. The predicted annual mean increment in NO_x concentrations is, however, too small (Table 4.10) to be expected to have a measurable effect on symptoms prevalence and long term respiratory health in the local community.

10.4 DISCUSSION AND CONCLUSIONS

Overall the risks to human health associated with SO₂ and NO_x emissions from glass furnaces are likely to be small, but under unfavourable weather conditions NO_x concentrations could exceed the Government's Air Quality Standards. Short term exposure to elevated concentrations of SO₂ or NO_x might cause some exacerbation of symptoms in asthmatics and others with pre-existing respiratory illness. Exceedence of the 1 hour air quality objective for NO_x may be associated with adverse respiratory effects in susceptible individuals.

In addition to their potential effects on human health, both SO₂ and NO_x are associated with the formation of acid rain, eutrophication of surface waters (nutrient excess leading to de-oxygenation of water bodies) and the formation of secondary particles. Sulphur dioxide is more strongly associated with acid rain and the formation of secondary particles than NO_x, whereas NO_x is more strongly associated with eutrophication and is also associated with the formation of low level ozone.

11. OVERALL ASSESSMENT OF HEALTH IMPACT LIKELY TO BE ASSOCIATED WITH PARTICLE EMISSIONS FROM GAS-FIRED GLASS FURNACES

Particle emissions from gas-fired glass furnaces are dominated by sodium sulphate. Concentrations of minor components of the waste gas dust including heavy metals and crystalline silica are far too small to be expected to have any association with adverse health effects. Dispersion modelling suggests that the long term increment in local concentrations of airborne particles associated with emissions from Pilkington's St Helens glassworks is extremely small. Under conditions of extremely poor dispersion the maximum increment in daily mean concentrations associated with emissions from glassworks is only likely to be about $3 \mu\text{g m}^{-3}$. This suggests that emissions from glassworks are not likely to contribute significantly to exceedences of the UK's air quality objective for airborne particles as PM_{10} . Dispersion modelling also suggests that gaseous emissions from Pilkington's St Helens glassworks are unlikely to have a measurable effect on annual mean concentrations of air pollution in the surrounding area. Under conditions of extremely poor dispersion, however, concentrations of sulphur dioxide or nitrogen dioxide may occasionally exceed short term air quality standards. This might cause some exacerbation of respiratory symptoms in some individuals, particularly those with asthma.

There is relatively little information available about the specific potential toxic effects of exposure to airborne sodium sulphate. The association between exposure to airborne particles, specifically ambient PM_{10} , and adverse health effects is well established but the cause is poorly understood. Ultrafine ($<0.1\mu\text{m}$) particles appear to be particularly associated with inflammation. Particles emitted from glass furnaces, while respirable, appear to contain a smaller ultrafine component than typical traffic emissions and therefore would be expected to be less active per unit mass than ambient PM_{10} . The results of experiments with animals and with human volunteers suggest that the toxicity of inhaled sulphate salts varies with their acidity. Fine particles of sodium sulphate are likely to be less biologically active than ammonium sulphate (a typical sulphate salt present in urban air) which in turn is less biologically active than pure sulphuric acid aerosol. Other toxicological studies have suggested that the transition metals content of both sulphate salts and of ambient urban PM_{10} may play an important role in promoting oxidative damage to the airways. The transition metals content of emissions from glass works is generally smaller than that of ambient PM_{10} and may also be less bioavailable as most of the metals present are locked up in glass spherules. In addition, some of the adsorbed organic species present on ambient soot particles may also promote an inflammatory response. Overall, it seems likely that primary particle emissions from gas-fired glass furnaces have a lower toxicity per unit mass than typical urban PM_{10} . A limited programme of *in vitro* toxicity testing may be helpful in understanding the relative toxicity of waste gas dust from glassworks, pure sodium sulphate and more typical ambient PM_{10} .

Epidemiological studies have suggested an association between ambient concentrations of airborne sulphate and a range of health endpoints. It seems likely, however, that sulphate in these studies is acting as a marker for some other more toxicologically relevant component of airborne particulate such as $\text{PM}_{2.5}$ or aerosol acidity. There is limited evidence of an association between aerosol acidity and both short and long term effects on health that is independent of the general association between particles and health. Unfortunately there have been no epidemiological studies that have specifically addressed the relative toxicity of the soluble salt versus soot components of urban PM_{10} . It is therefore difficult to estimate the relative risks of adverse health effects associated with exposure to sodium sulphate aerosol as opposed to ambient PM_{10} . Inhaled sodium sulphate particles are likely to grow within the

respiratory tract through hygroscopic absorption of moisture and will therefore not penetrate as deeply into the respiratory system as typical particles of ambient PM₁₀. Exposure to sodium sulphate and other soluble salts may therefore be more strongly associated with upper rather than lower respiratory symptoms.

Emissions of sulphur dioxide and other sulphur oxides from gas-fired glass furnaces are substantial in relation to those of primary particles. It seems probable that during the first few seconds to minutes following emission, a proportion of these emissions will condense with water in the waste gas stream to form sulphuric acid and other secondary sulphate species. The resultant annual average increment in local concentrations of aerosol acidity is probably small in comparison with that due to emissions of SO₂ from power stations and transport but measurable short term increments in local levels of aerosol acidity may arise under particularly unfavourable weather conditions. Ground level concentrations of aerosol acidity are, however, likely to be small in comparison to the levels of acidity associated with adverse effects in both epidemiological and experimental studies.

In summary, it is likely that the (primary) particle emissions from glass furnaces would be associated with a smaller risk to human health per unit mass concentration than typical ambient PM₁₀ in urban areas. Given the volume of sulphur dioxide emitted, it is likely that emissions from glass furnaces are associated with some formation of secondary aerosol including sulphuric acid. These secondary particles are likely to have a greater biological activity than the primary emissions but represent only a small increment on local aerosol concentrations.

12. ACKNOWLEDGEMENTS

This report is based on a study funded by Pilkington plc (IOM Contract 119-503). Simon Slade of Pilkington provided confidential information about the nature of waste gas dust from glass furnaces. Normal Wright of Pilkington undertook the dispersion modelling.

This report was peer reviewed within the IOM by Fintan Hurley, Alastair Robertson and Richard Cullen.

13. REFERENCES

- Abbey DE, Nishino N, McDonnell WF, Birchette RJ, Knutsen SF, Beeson WL, Yang JX. (1999). Long-term inhalable particles and other air pollutants related to mortality in nonsmokers. *American Journal of Respiratory and Critical Care Medicine*; 159: 373-382.
- Abbey DE, Burchette RJ, Knutsen SF, McDonnell WF, Lebowitz MD, Enright PL. (1998a). Long-term particulate and other air pollutants and lung function in nonsmokers. *American Journal of Respiratory and Critical Care Medicine*; 158: 289-298.
- Abbey DE, Nishino N, McDonnell WF. (1998b). Development of chronic productive cough associated with long-term ambient inhalable particulate pollutants (PM₁₀) in non-smoking adults: The AHSMOG study. *Applied Occupational and Environmental Hygiene*; 13: 444-452
- Alarie YC, Krumm AA, Busey WM, Ulrich CE, Krantz RJ. (1975). Long-term exposure to sulfur dioxide, sulfuric acid mist, fly ash, and their mixtures: results of studies in monkeys and guinea pigs. *Archives of Environmental Health*; 30: 254-262.
- Alarie C, Busey WM, Krumm AA and Ulrich CE. (1973). Long-term continuous exposure to sulfuric acid mist in cynomolgus monkeys and guinea pigs. *Archives of Environmental Health*; 27: 16-24.
- Amdur M. (1996). Animal toxicology. In: Wilson R, Spengler J, eds. *Particles in our air*. Cambridge, Massachusetts, Harvard University Press: 85-122.
- Amdur MO, Bayles J, Ugro V, Underhill DW. (1978). Comparative potency of sulfate salts. *Environmental Research*; 16: 1-8.
- American Thoracic Society. (1999). Health effects of outdoor air pollution: parts 1 and 2. *American Journal of Respiratory Care Medicine*; 153: 3-50; 477-498.
- Anderson HR, Spix C, Medina S, Schouten J, Castellsague J, Rossi G, Zmirou D, Touloumi G, Wojtyniak B, Vonk J, Bisanti L, Schwartz J, Katsouyanni K. (1997). Urban air pollution and emergency admissions for asthma in four European cities: the APHEA project. *Thorax*; 52: 760-765
- Anderson KR, Avol EL, Edwards SA, Shamo DA, Peng RC, Linn WS, Hackney JD. (1992). Controlled exposures of volunteers to respirable carbon and sulfuric acid aerosols. *Journal of Air and Waste Management Association*; 42: 770-776.
- Ansari AS, Pandis SN. (1999). Prediction of multicomponent inorganic atmospheric aerosol behaviour. *Atmospheric Environment*; 33: 745-757.
- Aranyi C, Vana SC, Thomas PT *et al.* (1983). Effects of subchronic exposure to a mixture of O₃, SO₂, and (NH₄)₂SO₄ on host defenses of mice. *Journal of Toxicology and Environmental Health*; 12: 55-71.
- Avol EL, Linn WS, Wightman LH, Whynot KR, Hackney JD. (1988). Short term respiratory effects of sulfuric acid in fog: a laboratory study of healthy and asthmatic volunteers. *Journal of the Air Pollution Control Association*; 38: 258-263.

- Beerkens NMR, Slad S, van Macke G, Tackels G. (1998). An examination of the low environmental impact of dust emitted by soda-lime glass furnaces. Unpublished glass industry document.
- Brauer M, Dumyahn TS, Spengler JD, Gutschmidt K, Heinrich J, Wichmann HE. (1995). Measurement of acidic aerosol species in Eastern Europe: implications for air pollution epidemiology. *Environmental Health Perspectives*; 103: 482-488.
- Burilkov T, Micahailova-Dotschewa L. (1990). Dangers of exposure to dust: extraction and production of natural gypsum. *Wiss-Umw -2*; 89-91.
- Burnett RT, Cakmak S, Brook JR, Krewski D. (1997). The role of particulate size and chemistry in the association between summertime ambient air pollution and hospitalization for cardiorespiratory diseases. *Environmental Health Perspectives*: 105; 614-620.
- Burnett RT, Dales R, Krewski D. (1995). Associations between ambient particulate sulfate and admissions to Ontario hospitals for cardiac and respiratory disease. *American Journal of Epidemiology*: 142; 15-22
- Burr ML, Anderson HR, Austin JB, Harkins LS, Kaur B, Strachan DP, Warner JO. (1999). Respiratory symptoms and home environment in children: a national survey. *Thorax*; 54: 27-32.
- Busch RH, Buschbom RL, Cannon WC, Lauhala KE, Miller FJ, Graham JA, Smith LG. (1984). Effects of ammonium sulfate aerosol exposure on lung structure of normal and elastase-impaired rats and guinea pigs. *Environmental Research*; 33: 454-472.
- Carter JD, Ghio AJ, Samet JM, Devlin RB. (1997). Cytokine production by human airway epithelial cells after exposure to an air pollution particle is metal-dependent. *Toxicological Pharmacology*; 146: 180-188.
- Charles JM, Menzel DB. (1975). Ammonium and sulfate ion release of histamine from lung fragments. *Archives of Environmental Health*; 30: 314-316.
- Chen LC, Fine JM, Qu Q-S, Amdur MO, Gordon T. (1992a). Effect of fine and ultrafine sulfate acid aerosols in guinea pigs: Alterations in alveolar macrophage function and intracellular pH. *Toxicology and Applied Pharmacology*; 113: 109-117.
- Chen LC, Miller PD, Amdur MO, Gordon T. (1992b). Airway hyperresponsiveness in guinea pigs exposed to acid-coated ultrafine particles. *Journal of Toxicology and Environmental Health*; 35: 165-174.
- Cloudhury AH, Gordian ME, Morris SS. (1997) Associations between respiratory illness and PM₁₀ air pollution. *Archives of Environmental Health*: 53; 113-117.
- Cocks AT and Fernando RP. (1982). The growth of sulphate aerosols in the human airways. *Journal of Aerosol Science*; 13: 9-19.
- COMEAP. (1995). Non-biological particles and health. Committee on the Medical Effects of Air Pollutants. Department of Health London: HMSO.
- COMEAP. (1998). Quantification of the effects of air pollution on health in the United Kingdom. Department of Health London: HMSO.

- Costa D. (1999) Role of metals. *Biology of Air Pollution*, 25th-25th October, 1999. London.
- Cullen RT, Tran CL, Buchanan D, Davis JMG, Donaldson K, Jones AD, Searl A. (1999). Investigations into the pulmonary effects of low toxicity dusts. Part I: Relative toxicological potency of dusts. (HSE Contract Research Report No. 216/1999). Sudbury: HSE Books.
- Culp DJ, Latchney LR, Frampton MW, Jahnke MR, Morrow PE, Utell MJ. (1995). Composition of human airway mucins and effects after inhalation of acid aerosol. *American Journal of Physiology*; 269: L358-L370.
- Delfino RJ, Murphy Moulton AM, Burnett RT, Brook JR, Becklake MR. (1997). Effects of air pollution on emergency room visits for respiratory illnesses in Montreal, Quebec. *American Journal of Respiratory and Critical Care Medicine*; 155: 568-576.
- Delfino RJ, Becklake MR, Hanley JA. (1994). The relationship of urgent hospital admissions for respiratory illnesses to photochemical air pollution levels in Montreal. *Environmental Research*; 67: 1-19.
- Department of the Environment. (1995). Processes prescribed for air pollution control by local enforcing authorities: Secretary of State's Guidance – Glass (excluding lead glass) manufacturing processes. PG3/3 (95)
- Department of the Environment, Transport and the Regions. (1999). The air quality strategy for England, Scotland, Wales and Northern Ireland. A consultation document. London: HMSO.
- Dockery DW, Cunningham J, Damokosh AI, Neas LM, Spengler JD, Koutrakis P, Ware JH, Raizenne M, Speizer FE. (1996). Health effects of acid aerosols on North American children: respiratory symptoms. *Environmental Health Perspectives*; 104: 500-505.
- Dockery DW, Pope CA III, Xu X, Spengler JD, Ware JH, Fay ME, Ferris BG Jr, Speizer FE (1993). An association between air pollution and mortality in six US cities. *New England Journal of Medicine*; 329: 1753-1759.
- Dockery DW, Schwartz J, Spengler JD. (1992). Air pollution and daily mortality: associations with particulates and acid aerosols. *Environmental Research*; 59: 362-373.
- Dockery DW, Speizer FE, Stam DO, Ware JH, Spengler JD, Ferris BG. (1989). Effects of inhalable particles on respiratory health of children. *American Review of Respiratory Disease*; 139: 587-594.
- Dohan FC, Everts GS, Smith R. (1962). Variations in air pollution and the incidence of respiratory disease. *Journal of Air Pollution Control Association*; 12: 418-436.
- Donaldson K, Stone V, MacNee W. (1999). The toxicology of ultrafine particles. In: Maynard RL, Howard CV, eds. *Particulate Matter: properties and effects upon health*. Oxford: Bios Scientific.
- Donaldson K, Brown DM, Mitchell C, Dineva M, Beswick PH, Gilmour P, MacNee W. (1997). Free radical activity of PM₁₀: Iron-mediated generation of hydroxyl radicals. *Environmental Health Perspectives*; 105: 1285-1289.

- Donaldson K, Bolton RE, Jones AD, Brown GM, Robertson MD, Slight J, Cowie H, Davis JMG. (1988). Kinetics of the bronchoalveolar leucocyte response in rats during exposure to equal airborne mass concentrations of quartz, chrysotile asbestos, or titanium dioxide. *Thorax*; 43: 525-533.
- Dreher KL, Jaskot RH, Lehmann JR, Richards JH, McGee JK, Ghio AJ, Coster DL. (1997). Soluble transition metals mediate residual oil fly ash induced acute lung injury. *Journal of Toxicology and Environmental Health*; 50: 285-305.
- Driscoll K E. (1996). Role of inflammation in the development of rat lung tumors in response to chronic particle exposure. *Inhalation Toxicology* 8 (Suppl):139-153.
- Drummond JG, Aranyi C, Schiff LJ, Fenters JD, Graham JA. (1986). Comparative study of various methods used for determining health effects of inhaled sulfates. *Environmental Research*; 41: 514-528.
- El-Fawal HAN and Schlesinger RB. (1994). Nonspecific airway hyperresponsiveness induced by inhalation exposure to sulfuric acid aerosol: An *in vitro* assessment. *Toxicology and Applied Pharmacology*; 152: 70-76.
- EPAQS. (1998). Lead. London: DETR.
- van der Eerden LJ. (1993). Risk of damage to vegetation caused by Na₂S₄ emissions from glass factories. IPO-DLO Report no. 93-03
- Evans J, Wolff S. (1996). Modelling of air pollution impacts: one possible explanation of the observed chronic mortality. In: Wilson R, Spengler J. *Particles in our air* (editors R Wilson and J Spengler), Harvard University Press: 189-204.
- Fine JM, Gordon T, Thompson JE, Sheppard D. (1987). The role of titratable acidity in acid aerosol induced bronchoconstriction. *American Review of Respiratory Disease*; 135: 826-830.
- Ferron GA. (1977). The size of soluble aerosol particles as a function of the humidity of the air. *Application to the human respiratory tract*; 8: 251-267.
- Folinsbee LJ. (1989). Human health effects of exposure to airborne acid. *Environmental Health Perspectives*; 79: 195-199.
- Frampton MW, Morrow PE, Cox C, Levy PC, Speers DM, Gibb FR, Condemi JJ, Utell MJ. (1995). Does pre-exposure to acidic aerosols alter airway responses to ozone in humans? *American Review of Respiratory Disease*; 145: A428
- Frampton MW, Morrow PE, Cox C, Levy PC, Condemi JJ, Speers D, Gibb FR, Utell MJ. (1995). Sulfuric acid aerosol followed by ozone exposure in healthy and asthmatic subjects. *Environmental Research*; 69: 1-14.
- Gavett SH, Madison SL, Dreher KL, Winsett DW, McGee JK, Costa DL. (1997). Metal and sulfate composition of residual oil fly ash determines airway hyperreactivity and lung injury in rats. *Environmental Research*; 72: 162-172.

- Gearhart JM, Schlesinger RB. (1988). Response of the tracheobronchial mucociliary clearance system to repeated irritant exposure: effect of sulfuric acid mist on function and structure. *Experimental Lung Research*; 14: 587-605
- Gilmour PS, Brown DM, Lindsay TG, Beswick PH, MacNee W and Donaldson K. (1996). Adverse health effects of PM₁₀ particles: involvement of iron in generation of hydroxyl radicals. *Occupational and Environmental Medicine*; 53: 817-822.
- Goldstein E, Chang DY, Lippert W, Tarkington B. (1979). Effect of near ambient exposures to sulfur dioxide and ferrous sulfate particles on murine pulmonary defense mechanisms. *Archives of Environmental Health*; 34: 424-31.
- Gomez SR, Parker RA, Dosman JA, McDuffie HH (1992) Respiratory health effects of alkali dust in residents near desiccated Old Wives Lake. *Archives of Environmental Health*; 47: 364-369.
- Gordian ME, Ozkaynak H, Xue J, Morris SS, Spengler. (1996). Particulate air pollution and respiratory disease in Anchorage, Alaska. *Environmental Health Perspectives*; 104: 290-297.
- Gupta A, Tang D, McMurry PH. (1995). Growth of monodisperse, submicron aerosol particles exposed to SO₂, H₂O and NH₃. *Journal of Atmospheric Chemistry*; 20: 117-139.
- Health and Safety Executive. (1999). Investigations into the pulmonary effects of low toxicity dusts. Parts 1 and 2. Sudbury: HSE (HSE Contract Research Report No 216/99).
- Hoek G, Brunekreef B. (1994). Effects of low-level winter air pollution concentrations on the respiratory health of Dutch children, *Environmental Research*; 64: 136-150.
- Hoek G, Brunekreef B. (1995). Effect of photochemical air pollution on acute respiratory symptoms in children. *American Journal of Respiratory Care Medicine*; 151: 27-32.
- Hoek G, Mennen Mg, Allen GA, Hofschreuder P, Van der Moulen T (1996) Concentrations of acidic air pollutants in the Netherlands. *Atmospheric Environment*; 30: 3141-3150.
- Holgate S. (1999). Pathogenic mechanisms in air pollution – induced lung injury. *Biology of Air Pollution*, 25th-25th October, 1999, London.
- Holma B. (1989). Effects on inhaled acids on airway mucus and its consequences for health. *Environmental Health Perspectives*; 79: 109-113.
- Ito K, Thurston GD. (1989). Characterization and reconstruction of historical London, England acid aerosol concentrations. *Environmental Health Perspectives*; 79: 35-42.
- Ito K, Thurston GD, Hayes C, Lippmann M (1993). Associations of London, England, daily mortality with particulate matter, sulfur dioxide and acidic aerosol pollution. *Archives of Environmental Health*; 48: 213-220.
- Jakab GJ, Clarke RW, Hemenway DR, Longphre MV, Kleeberger SR, Frank R. (1996). Inhalation of acid coated carbon black particles impairs alveolar macrophage phagocytosis. *Toxicological Letters*; 88: 243-248.
- Jedrychowski W, Krzyanowski M. (1989). Ventilatory lung function and chronic chest symptoms among the inhabitants of urban areas with various levels of acid aerosols: prospective study in Cracow. *Environmental Health Perspectives*; 79: 101-107.

Katsouyanni K, Touloumi G, Spix C, Schwartz J, Balducci F, Medina S, Rossie G, Wojtyniak B, Sunyer J, Bacharova L, Schouten JP, Ponka A, Anderson HR. (1997). Short term effects of ambient sulphur dioxide and particulate matter on mortality in 12 European cities: results from time series data from the APHEA project. *British Medical Journal*; 314: 1558-1563.

Kelada F, Evinton LE. (1978). Health effects of long term exposure to sodium sulfate dust. *Journal of Occupational Medicine*; 20: 812-814.

Kerminen VM, Pakkanen TA, Hillamo RE. (1997). Interactions between inorganic trace gases and supermicrometer particles at a coastal site. *Atmospheric Environment*; 31: 2753-2765

Kitabatake M, Imai M, Nakano H, Yoshida K. (1991). Effects of exposure to sulfate aerosols and antigen on breathing curve patterns of guinea pigs. *Journal of Toxicology and Environmental Health*; 3: 157-170.

Kinney PL, Thurston GD, Riazanne M. (1996). The effects of ambient ozone on lung function in children: a reanalysis of six summer camp studies. *Environmental Health Perspectives*; 104: 170-174.

Kitto AMN, Harrison RM. (1992). Processes affecting concentrations of aerosol strong acidity at sites in eastern England. *Atmospheric Environment. Part A. General Topics*; 26: 2389-2399.

Kittelson DB, Arnold M, Watts WE. (1999). Review of diesel particulate matter sampling methods. Final Report. University of Minnesota, Department of Mechanical Engineering.

Kleeman MJ Case GR. (1999). Effect of emissions control strategies on the size and composition distribution of urban particulate air pollution. *Environmental Science and Technology*; 33: 177-189.

Kleinman MT, Bailey RM. (1985). Controlled exposure to a mixture of sulphur dioxide, nitrogen dioxide and particulate air pollutants: effects on human pulmonary function and respiratory symptoms. *Archives of Environmental Health*; 40: 197-201.

Kleinman MT, Phalen RF, Mautz WJ, Mannix RC, McClure TR, Crocker TT. (1989). Health effects of acid aerosols formed by atmospheric mixtures. *Environmental Health Perspectives*; 79: 137-45.

Kleinman MT, Linn WS, Bailey RM, Anderson KR, Medway DA, Hackney JD (1981) Human exposure to ferric sulfate aerosol: effects on pulmonary function and respiratory symptoms. *American Industrial Hygiene Association Journal*; 42: 298-304.

Koenig JQ, Covert DS, Larson TV, Pierson WE. (1993a). The effect of duration of exposure on sulfuric acid-induced pulmonary function changes in asthmatic adolescent subjects: a dose response study. *American Review of Respiratory Disease*; 145: A428.

Koenig JQ, Dumler K, Rebolledo V, Williams PV, Pierson WE. (1993b). Respiratory effects of inhaled sulfuric acid on senior asthmatics and nonasthmatics. *Archives of Environmental Health*; 48: 171-175.

Koenig JQ, Covert DS, Pierson WE. (1989). Effects of inhalation of acidic compounds on pulmonary function in allergic adolescent subjects. *Environmental Health Perspectives*; 79: 173-178

- Koenig JQ, Pierson WE, Horike M, Frank R. (1981). Effects of SO₂ plus NaCl aerosol combined with moderate exercise on pulmonary function in asthmatic adolescents. *Environmental Research*; 25: 340-348.
- Koenig JQ, Pierson WE, Frank R. (1980). Acute effects of inhaled SO₂ plus NaCl droplet aerosol on pulmonary function in asthmatic adolescents. *Environmental Research*; 22: 145-153.
- Korrick SA, Neas LM, Dockery DW, Gold DR, Allen GA, Hill LB, Kimball KD, Rosner BA, Speizer FE. (1998). Effects of ozone and other pollutants on the pulmonary function of adult hikers. *Environmental Health Perspectives*; 106: 93-99.
- Koutrakis P, Wolfson JM, Spengler JD. (1989). Equilibrium size of atmospheric aerosol sulfates as acidity and ambient relative humidity. *Journal of Geophysical Research*; 94: 6442-6448.
- Larson TV, Frank R, Covert DS, Holub D, Morgan MS. (1982). Measurement of respiratory ammonia and the chemical neutralisation of inhaled sulfuric acid aerosol in anaesthetized dogs. *American Review of Respiratory Disease*; 125: 502-506.
- Last JA. (1991). Global Atmospheric change: Potential health effects of acid aerosol and oxidant gas mixtures. *Environmental Health Perspectives*; 96: 151-157.
- Last JA, Hyde DM, Chang DP. (1984). A mechanism of synergistic lung damage by ozone and a respirable aerosol. *Experimental Lung Research*; 7: 223-35.
- Lazaridis M, Melas D. (1998). New sulfate particle formation during summer pollution episodes using a 3D aerosol model. *Journal of Aerosol Science*; 29: 913-927.
- Lazaridis M, Koutrakis P. (1997). Simulation of formation and growth of atmospheric sulfate particles. *Journal of Aerosol Science*; 28: 107-119.
- Leduc D, Fally S, De Vuyst P, Wollast R, Yernault JC. (1996). Acute exposure to realistic acid fog: effects on respiratory function and airway responsiveness in asthmatics. *Environmental Research*; 71: 89-98.
- Lee DS, Dollard GJ, Derwent RG, Pepler S. (1999). Observations on gaseous and aerosol components of the atmosphere and their relationships. *Water Air and Soil Pollution*; 113: 175-202
- Linn WS, Gong H, Shamoo DA, Anderson KR, Avol EL. (1997). Chamber exposures of children to mixed ozone, sulfur dioxide and sulfuric acid. *Archives of Environmental Health*; 52: 179-187
- Linn WS, Anderson KR, Shamoo DA, Edwards SA, Webb TL, Hackney JD, Gong H Jr. (1995). Controlled exposures of young asthmatics to mixed oxidant gases and acid aerosol. *American Journal of Respiratory and Critical Care Medicine*; 152: 885-891.
- Linn WS, Shamoo DA, Anderson KR, Peng RC, Avol EL, Hackney JD. (1994). Effects of prolonged, repeated exposure to ozone, sulfuric acid and their combination in health and asthmatic volunteers. *American Journal of Respiratory and Critical Care Medicine*; 150: 431-440.

Linn WS, Shamoo DA, Anderson KR, Peng RC, Avol EL, Hackney JD. (1993). Responses of asthmatics in prolonged, repeated exposures to ozone and sulfuric acid: comparison with healthy subjects. *American Review of Respiratory Disease*; 145: A428.

Lipfert FW. (1994). *Air pollution and community health: a critical review and data sourcebook*. New York: Van Nostuand Reinhold.

Lippmann M, Ito K. (1995). Separating the effects of temperature and season on daily mortality from those of air pollution in London: 1965-1972. *Inhalation Toxicology*; 7: 85-97.

Lippmann M, Thurston GD. (1996). Sulfate concentrations as an indicator of ambient particulate matter air pollution for health risk evaluations. *Journal of Exposure Analysis and Environmental Epidemiology*; 6: 123-146.

Loscutoff SM, Cannon WC, Buschbom RL, Busch RH, Killand BW. (1985). Pulmonary function in elastase-treated guinea pigs and rats exposed to ammonium sulfate or ammonium nitrate aerosols. *Environmental Research*; 36: 170-80.

MAAPE. (1991). *Ozone. Advisory Group on the Medical Aspects of Air Pollution Episodes*, (Department of Health, 1st report). London: HMSO.

MAPPE. (1992). *Sulphur dioxide, acidic aerosols and particles. Advisory Group on the Medical Aspects of Air Pollution Episodes*. (Department of Health, 2nd report). London: HMSO.

MAAPE. (1993). *Oxides of nitrogen. Advisory Group on the Medical Aspects of Air Pollution Episodes*. (Department of Health, 3rd report). London: HMSO.

MAPPE. (1995). *Health effects of exposures to mixtures of air pollutants. Advisory Group on the Medical Aspects of Air Pollution Episodes*. (Department of Health, 4th report). London: HMSO.

Meng Z, Seinfeld JH. (1994). On the source of the submicrometer droplet mode of urban and regional aerosols. *Aerosol Science and Technology*; 20: 253-265.

McConnell R, Berhane K, Gilliland F, London SJ, Vora H, Avol E, Gauderman WJ, Margolis HG, Lurmann F, Thomas DC, Peters JM. (1999). Air pollution and bronchitic symptoms in Southern California children with asthma. *Environmental Health Perspectives*; 107: 757-760.

Micklin PP. (1988). Desiccation of the Aral Sea: A water management disaster in the Soviet Union. *Science*; 241: 1170-76.

Morgan MS, Koenig J, Covert DS, Frank R. (1977). Acute effects of inhaled SO₂ combined with hygroscopic aerosol in healthy man. *American Review of Respiratory Disease*; 111: 763-773.

Muller B, Seifart C, Barth PJ. (1998). Effect of air pollutants on pulmonary surfactant system. *European Journal of Clinical Investigation*; 28: 762-777.

Naether LP, Hoford TR, Beckett WS, Belanger K, Triche EW, Bracken MB, Leaderer BP (1999). Healthy women's PEF variations with ambient summer concentrations of PM₁₀, PM_{2.5}, SO₄²⁻, H⁺ and O₃. *American Journal of Respiratory and Critical Care Medicine*; 160: 117-125.

- Neas LM, Dockery DW, Koutrakis P, Speizer FE. (1999). Fine particles and peak flow in children: acidity versus mass. *Epidemiology*; 10: 550-553.
- Neas LM, Dockery DW, Burge H, Koutrakis P, Speizer FE. (1996). Fungus spores, air pollutants, and other determinants of peak expiratory flow rate in children. *American Journal of Epidemiology*; 143: 797-807.
- Neas LM, Dockery DW, Koutrakis P, Tollerud DJ, Speizer FE. (1995). The association of ambient air pollution with twice daily expiratory flow rate measurements in children. *American Journal of Epidemiology*; 141: 111-122.
- Nemery B. (1990). Metal toxicity and the respiratory tract. *European Respiratory Journal*; 3: 202-219.
- NETCEN. (1998). Air pollution in the UK: 1996. Report prepared for the DETR. AEA/RAMP/20441002/001.
- Oberdorster G. (1996). Significance of particle parameters in the evaluation of exposure-dose-response relationships of inhaled particles. *Inhalation Toxicology*; 8 (supplement): 73-89.
- Ostro BD. (1990). Associations between morbidity and alternative measures of particulate matter. *Risk Analysis*; 10: 421-427.
- Ostro BD, Lipsett MJ, Wiener MB, Selner JC. (1991). Asthmatic responses to airborne acid aerosols. *American Journal of Public Health*; 81: 694-702.
- Ozkaynak H, Xue J, Zhou H, Spengler D, Thurston GD. (1996). Intercommunity differences in acid aerosol (H^+)/sulfate (SO_4) ratios. *Journal of Exposure Analysis and Environmental Epidemiology*; 6: 35-55.
- Pepelko WE, Mattox JK, Cohen AL. (1980). Toxicology of ammonium sulfate in the lung. *Bulletin of Environmental Contaminant Toxicology*; 24: 156-60.
- Peters A, Wichmann HE, Tuch T, Heinrich J, Heyder J. (1997a). Respirable effects are associated with the number of ultrafine particles. *American Journal of Respiratory and Critical Care Medicine*; 155: 1376-1383.
- Peters A, Dockery DW, Heinrich J, Wichmann HE. (1997b). Short-term effects of particulate air pollution on respiratory morbidity in asthmatic children. *European Respiratory Journal*; 10: 872-879.
- Peters JM, Avol E, Gauderman WJ, Linn WS, Navidi W, London SJ, Mergolis H, Rappaport E, Vora H, Gong H, Thomas DC. (1999a). A study of twelve southern California communities with differing levels and types of air pollution II Effects on pulmonary function. *American Journal of Respiratory and Critical Care Medicine*; 159: 768-775.
- Peters JM, Avol E, Navidi W, London SJ, Gauderman WJ, Lurmann F, Linn WS, Mergolis H, Rappaport E, Gong H, Thomas DC. (1999b). A study of twelve southern California communities with differing levels and types of air pollution I Prevalence of respiratory morbidity. *American Journal of Respiratory and Critical Care Medicine*; 159: 760-767.
- Phalen RF, Kenoyer JL, Crocker TT, McClure TR. (1980). Effects of sulfate aerosols in combination with ozone on elimination of tracer particles inhaled by rats. *Journal of Toxicology and Environmental Health*; 6: 797-810.

- Poloneiki JD, Atkinson RW, Ponce de Leon A, Anderson HR. (1997). Daily time series for cardiovascular hospital admissions and previous day's air pollution in London, UK. *Occupational and Environmental Medicine*; 54: 535-540.
- Pope A, Dockery D (1996) Epidemiology of chronic health effects: cross sectional studies. In: Wilson R, Spengler J, eds. *Particles in our air*. Cambridge, Massachusetts: Harvard University Press: 149-168.
- Pope CA, Dockery DW. (1992). Acute health effects of PM₁₀ pollution on symptomatic and asymptomatic children. *American Review of Respiratory Disease*; 145: 1123-1128.
- Pope CA, Dockery DW, Schwartz J. (1995a). Review of epidemiological evidence of health effects of particulate air pollution. *Inhalation Toxicology*; 7: 1-18.
- Pope CA, Thun MJ, Namboodiri M, Dockery DW, Evans JS, Speizer FE, Heath CW. (1995b). Particulate air pollution as a predictor of mortality in a prospective study of US adults. *American Journal of Respiratory and Critical Care Medicine*; 151: 669-74.
- Pope CA, Schwartz J, Ransom MR. (1992). Daily mortality and PM₁₀ pollution in Utah valley. *Archives of Environmental Health*; 47: 211-217.
- Pope CA, Dockery DW, Spengler JD, Riazanne ME. (1991). Respiratory health and PM₁₀ pollution: a daily time series analysis. *American Review of Respiratory Disease*; 144: 668-674.
- Potukuchi S, Wexler AS. (1995). Identifying solid-aqueous phase transitions in atmospheric aerosols - I Neutral acidity solutions. *Atmospheric Environment*; 29: 1663-1676.
- Quality of Urban Air Review Group (QUARG). (1996). Airborne particulate matter in the United Kingdom. Quality of Urban Air Review Group, 3rd Report. Department of the Environment.
- Quality of Urban Air Review Group (QUARG). (1993). Urban air quality in the United Kingdom. Quality of Urban Air Review Group, 1st Report, Department of the Environment.
- Quay JL, Reed W, Samet J, Devlin RB. (1998). Air pollution particles induce IL-6 gene expression in human airway epithelial cells via NF-κB activation. *American Journal of Respiratory Cell Molecular Biology*; 19: 98-106.
- Quinn TL, Ondov JM. (1998). Influence of temporal changes in relative humidity on dry deposition velocities and fluxes of aerosol particles bearing trace elements. *Atmospheric Environment*; 32: 3467-3479.
- Raizenne M, Neas LM, Damokosh AI, Dockery DW, Spengler JD, Koutrakis P, Ware JH, Speizer FE. (1996). Health effects of acid aerosols on North American children: pulmonary function. *Environmental Health Perspectives*; 104: 506-514.
- Riazenne ME, Burnett RT, Stern B, Franklin CA, Spengler JD. (1989). Acute lung function responses to ambient acid aerosol exposures in children. *Environmental Health Perspectives*; 79: 179-185.
- Richards R. (1999). Bioreactivity and fate of carbonaceous particles. *Biology of Air Pollution*, 25th-25th October, 1999. London.

Russell LM, Seinfeld JH. (1998). Size- and composition-resolved externally mixed aerosol model. *Aerosol Science and Technology*; 28: 403-416.

Sackner MA, Dougherty RL, Chapman CA, Ciple J, Perez D, Kwoka M, Reinhart M, Brito M. (1981). Effects of brief and intermediate exposures to sulfate submicron aerosols and sulfate injections on cardiopulmonary function of dogs and tracheal mucous velocity of sheep. *Journal of Toxicology and Environmental Health*; 7: 951-972.

Sarangapani R, Wexler AS. (1996). Growth and neutralization of sulphate aerosols in human airways. *The American Physiological Society*; 81(1): 480-490.

Schlesinger RB. (1989). Factors affecting the response of lung clearance systems to acid aerosols: role of exposure concentration, exposure time, and relative acidity. *Environmental Health Perspectives*; 79: 121-126.

Schlesinger RB. (1983). Alteration of lung defense by acid sulfates. 76th Annual Meeting of the Air Pollution Control Association. Paper 83-3.4.

Schlesinger RB, Chen LC, Finkelstein I, Zelikoff JT. (1990). Comparative potency of inhaled acidic sulfates: speciation and the role of hydrogen ion. *Environmental Research*; 52: 210-24.

Schlesinger RB, Fine JM, Chen LC. (1992). Interspecies differences in the phagocytic activity of pulmonary macrophages subjected to acidic challenge. *Fundamental and Applied Toxicology*; 19: 584-9.

Schlesinger R, Lippmann M, Albert RE. (1978). Effects of short-term exposures to sulfuric acid and ammonium sulfate aerosols upon bronchial airway function. *American Industrial Hygiene Association Journal*; 39: 275-286.

Schwartz J, Norris G, Larson T, Sheppard L, Laiborne C, Koenig J. (1999). Episodes of high coarse particle concentrations are not associated with increased mortality. *Environmental Health Perspectives*; 107: 339-342.

Schwartz J, Dockery DW, Neas LM. (1996). Is daily mortality associated specifically with fine particles? *Journal of the Air and Waste Management Association*; 46: 927-939.

Schwartz J, Dockery DW, Neas LM, Wypij D, Ware JH, Spengler JD, Koutrakis P, Speizer FE, Ferris BG Jr. (1994). Acute effects of summer air pollution on respiratory symptom reporting in children. *American Journal of Respiratory and Critical Care Medicine*; 150: 1234-1242.

Searl A, Buchanan D. (2000). Measurement of the durability of man-made vitreous fibres. IOM Research Report TM/00/03.

Skornik WA, Brain JD. (1983). Relative toxicity of inhaled metal sulfate salts for pulmonary macrophages. *American Review of Respiratory Disease*; 128: 297-303.

Slade SJ. (2000). Characterisation of dust emissions from gas-fired flat glass furnaces; a non-confidential report. (Pilkington Internal Research Report TR/00/07).

Slade SJ (1999) An analysis of waste gas dust from V1 furnace, and its implications for Pilkington's emission control Strategy. (Pilkington Internal Research Report TR/99/53).

Slade SJ. (1998a). Initial analysis of waste gas dust from a rolled plate furnace. (Pilkington Internal Research Report TR/98/01).

Slade SJ. (1998b). Composition of waste gas dust from container furnaces (United Glass Peasley). (Pilkington Internal Research Report TR/98/04).

Slade SJ. (1998c). Indication that flat glass furnace waste gas dust emissions have no negative environmental impact, based on extensive analyses of dust emitted (includes Summary analysis of flat glass furnace waste gas dust emissions) (Pilkington Internal Research Report TR/98/18).

Slade SJ. (1998d). Investigation of dust deposition from St. Helens glass furnaces and implications for Pilkington's pollution control strategy. (Pilkington Internal Research Report TR/98/36).

Slade SJ. (1997a). An assessment of factors affecting particulate formation and emission by furnaces firing gas and oil fuels – Part 3: Collection and initial analysis of dust collected from a gas-fired furnace. (Pilkington Internal Research Report GR/97/70).

Slade SJ. (1997b). An assessment of factors affecting particulate formation and emission by furnaces firing gas and oil fuels – Part 4: Detailed analysis of dust collected from a gas-fired furnace and environmental impact implications. (Pilkington Internal Research Report GR/97/71).

Slade SJ. (1996). An assessment of factors affecting particulate formation and emission by furnaces firing gas and oil fuels – Part 2: Sodium sulphate form and particle morphology. (Pilkington Internal Research Report GR/96/64).

Slade SJ. (1995). An assessment of factors affecting particulate formation and emission by furnaces firing gas and oil fuels – Part 1: Furnace Na:S ratio, fuel H:C ratio and NaCl involvement. (Pilkington Internal Research Report GR/95/04).

Speizer FE. (1989). Studies of acid aerosols in six cities and in a new multi-city investigation: design issues. *Environmental Health Perspectives*; 79: 61-67.

Spektor DM, Yen BM, Lippmann N. (1989). Effect of concentration and cumulative exposure of inhaled sulfuric acid on tracheobronchial particle clearance in healthy humans. *Environmental Health Perspectives*; 79: 167-172.

Spengler JD, Koutrakis P, Dockery DW, Raizenne M, Speizer FE. (1996). Health effects of acid aerosols on North American children: air pollution exposures. *Environmental Health Perspectives*; 104: 492-499.

Spengler JD, Keeler GJ, Koutrakis P, Ryan PB, Raizenne M, Franklin CA. (1989). Exposures to acidic aerosols. *Environmental Health Perspectives*; 79: 48-51.

Spix C, Anderson NR, Schwartz J, Vigotti MA, Le Tertre A, Vonk JM, Touloumi G, Balducci F, Pedkarski T, Bacharova L, Tobias A, Panka A, Katsouyanni K. (1998). Short-term effects of air pollution on hospital admissions of respiratory diseases in Europe: a quantitative summary of APHEA study results. *Archives of Environmental Health*; 53: 54-64.

- Studnicka ME, Frischer T, Meinert R, Studnicka-Benke A, Hajek K, Spengler JD, Neumann MG. (1995). Acidic particles and lung function in children: a summer camp study in the Austrian Alps. *American Journal of Respiratory and Critical Care Medicine*; 151: 423-30.
- Stern BR, Raizenne ME, Burnett RT, Jones L, Kearney J, Franklin CA. (1994). Air pollution and childhood respiratory health: exposure to sulfate and ozone in 10 Canadian rural communities. *Environmental Research*; 66: 125-142.
- Stieb DM, Burnett RT, Beveridge RC, Brook JR. (1996). Association between ozone and asthma emergency department visits in Saint John, New Brunswick, Canada. *Environmental Health Perspectives*; 104: 1354-1360.
- Stohs SJ and Bagchi D. (1995). Oxidative mechanisms in the toxicity of metal ions. *Free Radical Biological Medicine*; 2: 321-336.
- Sunyer J, Spix C, Quenel P, Pnce-de-Leon, Barumandzadeh T, Touloumi G, Bacharova L, Wojtymiak B, Vonk J, Bisanti L, Schwarz J. (1997). Urban air pollution and emergency admissions for asthma in four European cities: The APHEA project. *Thorax*; 52: 760-765.
- Tarchi M, Orsi D, Comba P, Se Santis M, Pirastu R, Battista G, Valiani M. (1994). Cohort mortality study of rock salt workers in Italy. *American Journal of Industrial Medicine*; 25: 251-256.
- Thurston GD, Lipmann M, Scott MR, Samet JM. (1997). Summertime haze air pollution and children with asthma. *American Journal of Respiratory and Critical Care Medicine*; 155: 654-660.
- Thurston GD, Ito K, Hayes CG, Bates DV, Lippmann M. (1994). Respiratory hospital admissions and summertime haze air pollution in Toronto, Ontario: consideration of the role of acid aerosols. *Environmental Research*; 65: 271-290.
- Thurston GD, Ito K, Kinney PL, Lippmann M. (1992). A multi-year study of air pollution and respiratory hospital admissions in three New York State metropolitan areas: results for 1988 and 1989 summers. *Journal of Exposure Analysis and Environmental Epidemiology*; 2: 429-450.
- Touloumi G, Katsouyanni K, Zmirou D, Schwartz J, Spix C, De Leon AP, Tobias A, Quenel P, Rabczenko D, Bacharova L, Bisanti L, Vonk JM, Ponka A. (1997). Short term effects of ambient oxidant exposure on mortality: a combined analysis within the APHEA project. *American Journal of Epidemiology*; 146: 177-185.
- Utell M, Samet J. (1996). Airborne particles and respiratory disease: clinical and pathogenic considerations. In: Wilson R, Spengler J, eds. *Particles in our air*. Cambridge, Massachusetts, Harvard University Press: 169-188.
- Utell MJ, Frampton MW, Morrow PE, Cox C, Levy PC, Speers DM, Gibb FR. (1994). Effects of sequential sulfuric acid and ozone exposure on the pulmonary function of healthy subjects and subjects with asthma. *Health Effects Institute Report*; 70: 37-93.
- Utell MJ, Mariglio JA, Morrow PE, Gibb FR, Speers DM. (1989). Effects of inhaled acid aerosols on respiratory function: the role of endogenous ammonia. *Journal of Aerosol Medicine*; 2: 141-147.

- Utell MJ, Morrow PE, Hyde RW. (1984). Airway reactivity to sulfate and sulfuric acid aerosols in normal and asthmatic subjects. *Journal of the Air Pollution Control Association*; 34: 931-935.
- Utell MJ, Morrow PE, Speers DM, Darling J, Hyde RW. (1983). Airways responses to sulfate and sulfuric acid aerosols in asthmatics, an exposure response relationship. *American Review Respiratory Disease*; 128: 144-450.
- Utell MJ, Morrow PE, Hyde RW. (1982). Comparison of normal and asthmatic subjects' responses to sulphate pollutant aerosols. *Annals of Occupational Hygiene*; 26: 691-697.
- Utell MJ, Aquilina AT, Hall WJ, Speers DM, Douglas RG, Gibb FR, Morrow PE, Hyde RW. (1980). Development of airway reactivity to nitrates in subjects with influenza. *American Review of Respiratory Disease*; 121: 233-241.
- Utell MJ, Swinburne AJ, Hyde RW, Speers DM, Gibb RF, Morrow RE. (1979). Airway reactivity to nitrates in normal and mild asthmatic subjects. *Journal of Applied Physiology*; 46: 189-196.
- Wordley J, Walters S, Ayres JG. (1997). Short term variations in hospital admissions and mortality and particulate air pollution. *Occupational and Environmental Medicine*; 54: 108-116.
- World Health Organisation. (1998). Copper. International Programme on Chemical Safety, Environmental Health Criteria 200; Geneva: WHO.
- World Health Organisation. (1997). Nitrogen Oxides. International Programme on Chemical Safety, Environmental Health Criteria 188 (2nd ed). Geneva: WHO.
- World Health Organisation. (1995). Lead. International Programme on Chemical Safety, Environmental Health Criteria 165. Geneva: WHO.
- World Health Organisation. (1988a). Chromium. International Programme on Chemical Safety, Environmental Health Criteria 61. Geneva: WHO.
- World Health Organisation. (1988b). Vanadium. International Programme on Chemical Safety, Environmental Health Criteria 81. Geneva: WHO.
- World Health Organisation. (1981). Arsenic. International Programme on Chemical Safety, Environmental Health Criteria 18. Geneva: WHO.
- Wright N. (2000). St Helens pollution dispersion (XIX). Internal Pilkington Memo. ML/00/75
- Wyzga RE, Folinsbee LJ. (1995). Health effects of acid aerosols. *Water, Air and Soil Pollution*; 85; 177-188.
- Yu CP. (1996). Extrapolation modelling of particle deposition and retention from rats to humans. *Inhalation Toxicology*; 8 (suppl): 279-291.
- Zelikoff JT, Sisco MP, Yang Z, Cohen MD, Schlesinger RB. (1994). Immunotoxicity of sulfuric acid aerosol: effects on pulmonary macrophage effector and function activities critical for maintaining host resistance against infections diseases. *Toxicology*; 92: 269-286.

Zemp E, Elsasser S, Schindler C, Kunzli N, Perruchoud AP, Domenighetti G, Medici Tullio, U Ackermann-Liebrich, Leuenberger P, Monn C, Bolognini G, Bonagrad JP, Brandli O, Karrer W, Keller R, Schoni MH, Tschopp JM, Villiger B, Zellweger JP. (1999). Long term ambient air pollution and respiratory symptoms in adults (SAPALDIA Study). *American Journal of Respiratory and Critical Care Medicine*; 159: 1257-1266.

14. GLOSSARY

Airways reactivity: the extent to which the airways reacts to stimuli such as cold air, histamine, carbachol and acetylcholine that cause bronchoconstriction

Asthma: A chronic respiratory disease in which the airways are unusually sensitive to a range of stimuli. This results in episodic airway obstruction.

Atheroma: development of deposits lining blood vessels (atheromatosis – atheratous conditions of the arteries)

Alveoli: unciliated airways of the lung where gas exchange occurs

Bronchiolitis: bronchitis affecting the finest bronchial tubes.

Bronchitis: inflammation of bronchial tubes leading to excess mucous production and narrowing of the airways.

Bronchoconstriction: narrowing of the airways associated with conditions such as asthma

(Lung/ pulmonary) Clearance: processes by which inhaled particles are removed from the lung

COMEAP: Department of Health's Committee on the Medical Effects of Air Pollution

COPD: Chronic obstructive pulmonary disease

Cohort study: a study in which the fate of individual members of a cohort is examined. Studies may be prospective or retrospective.

Cross sectional study: an epidemiological study in which health effects are examined in different individuals or population groups at a single instant in time. In studies of ambient air pollution cross sectional studies are usually population-based, ie they involve comparison of populations in areas of differing air quality rather than comparison of individuals.

Cumulative exposure: concentration x time

DETR: Department of the Environment, Transport and the Regions

DoH: Department of Health

Dose-response function: function linking exposure to an agent to response. In studies of ambient air pollution exposure is usually described in terms of simple concentration, whereas in the workplace exposure is usually described as a function of concentration and duration of exposure.

Emphysema: degradation of air cells in lungs giving rise to large continuous air sacs causing breathlessness

(Airways) Epithelium: layer of cells lining the airways

EPAQS: Department of the Environment, Transport and the Region's Expert Panel for Air Quality Standards

ETS: Environmental tobacco smoke

FEV₁: The volume of air expired during the first second of a maximal or “forced” expiration

Fibrinogen: soluble protein of globulin class which occurs in blood plasma and is converted to an insoluble protein, fibrin, during the clotting process

Fibrosis: formation of scar tissue

FVC: Forced vital capacity. The volume of air expired in a forced expiration following maximum inspiration

Longitudinal study: an epidemiological study in which the fate of individuals or a population is tracked through time.

MAAPE: Department of Health’s Advisory Group on the Medical Aspects of Air Pollution Episodes, Department of Health

MMEF: Maximal mid-expiratory flow

(Alveolar) Macrophages: specialist cells that protect the surface of the lung and among other functions engulf and remove inhaled particles

Odds Ratio: Ratio of risk of disease relative to different levels of exposure in a case-control epidemiological study (ie individuals were included in the study if they had/ did not have a particular disease endpoint of interest).The odds ratio is approximately the same as the relative risk, if the outcome of interest is rare.

Phagocytosis: engulfment of foreign particles by a specialist cells (macrophages in the lung)

Prospective study: the fate of cohort members is followed following their recruitment into the study.

QUARG: Quality of Urban Air Review Group

Relative Risk: ratio of the risk of disease in exposed population relative to the risk in an unexposed population

Retrospective study: a study in which the exposure history of a cohort is estimated from historical records

sRAW: specific airways resistance – the product of the airway resistance and the lung volume at which the measurement was made.

Thrombin: albumin-like protein having a very powerful clotting activity when added to whole blood. Not present in unclotted circulating blood.

TSP: total suspended particulate

Applying science for a better working environment

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The IOM is a major independent centre of scientific excellence in the fields of occupational and environmental health, hygiene and safety. We aim to provide quality research, consultancy and training to help to ensure that people's health is not damaged by conditions at work or in the environment. Our principal research disciplines are exposure assessment, epidemiology, toxicology, ergonomics and behavioural and social sciences, with a strong focus on multi-disciplinary approaches to problem solving.

Our beginnings

Our first major research programme began in the 1950s, on respiratory health problems in the coal mining industry. Major themes were quantification of airborne dust concentrations in different jobs, characterisation of types and constituents of the dusts, measurement of health effects, relationships between exposure and disease, and proposals for prevention. This research became an international benchmark for epidemiological studies of occupational health, and was the primary influence on dust standards in mines in the UK, US and other countries.

Current themes

Our current work spans many other industries including asbestos, MMMF, pesticides, chemicals, energy, telecoms, metals, textiles, construction, agriculture as well as the environment. While diseases of the respiratory tract remain a major interest, our scope now extends to many other health outcomes such as mortality, cardiovascular effects, cancer, back pain, upper-limb disorders, hearing loss, skin diseases, thermal stress and psychological stress. Related work includes the development and application of measurement and control systems, mathematical models and survey methods.

Who we work for

Our work in these areas is conducted for a wide range of organisations in the UK, the EU, and the US, including Government departments, international agencies, industry associations, local authorities, charitable organisations, and industrial and commercial companies. The IOM is a World Health Organisation (WHO) collaborating centre and is an approved institute of the Universities of Edinburgh and Aberdeen, enjoying collaborative research links with NIOSH, IARC, and many other institutes throughout the world.

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