ISBN 92 890 1114 9
ISSN 0378-2255

PRINTED IN DENMARK

CONTENTS

ISBN 92 890 1114 9
ISSN 0378-2255

PRINTED IN DENMARK

CONTENTS

30

Sulfur dioxide and particulate matter

General Description

Sulfur dioxide (SO_2) and particles derived from the combustion of fossil fuels are major air pollutants in urban areas of the world. Sulfur oxides (SO_x) and particulate matter are parts of a complex pollutant mixture. For guideline purposes, a division into three categories is appropriate:

(*a*) sulfur dioxide,

(*b*) the acid aerosols that may result from the oxidation of sulfur dioxide in the atmosphere, and

(*c*) sulfur dioxide plus particles.

Sulfur dioxide. Sulfur dioxide is a colourless gas that reacts on the surface of a variety of airborne solid particles. It is readily soluble in water and can be oxidized within airborne water droplets.

Sulfur dioxide results from the combustion of sulfur-containing fossil fuels, the smelting of sulfur-containing ores, and other industrial processes. Domestic fires can also produce emissions containing sulfur dioxide.

Acid aerosol. Sulfuric acid (H_2SO_4) is a strong acid that is formed from the reaction of sulfur trioxide gas (SO_3) with water. Sulfuric acid is strongly hygroscopic. As a pure material, it is a clear colourless liquid with a boiling-point of 330 °C. Ammonium bisulfate (NH_4HSO_4), which is less acidic than sulfuric acid as a pure material, is a crystalline solid, with a melting-point of 147 °C.

Particulate matter. Airborne particulate matter represents a complex mixture of organic and inorganic substances. Mass and composition tend to divide into two principal groups: coarse particles larger than 2.5 μm in aerodynamic diameter, and fine particles smaller than 2.5 μm in aerodynamic diameter. The smaller particles contain the secondarily formed

aerosols (gas to particle conversion), combustion particles and recondensed organic and metal vapours. The larger particles usually contain earth crustal materials and fugitive dust from roads and industries. The acid component of particulate matter, and most of its mutagenic activity, is generally contained in the fine fraction, although in fog some coarse acid droplets are also present.

Because of the complexity of particulate matter and the importance of particle size in determining exposure, multiple terms are used to describe particulate matter. Some terms are derived from and defined by sampling methods, e.g. suspended particulate matter, total suspended particulates, black smoke. Other terms refer more to the site of deposition in the respiratory tract, e.g. inhalable, thoracic particles that deposit primarily in the lower respiratory tract below the larynx. Other terms, such as PM_{10} (particulate matter with an aerodynamic diameter of 10 μm), have both physiological and sampling components.

Methods for sampling and analysing suspended particulate matter were discussed by WHO *(1)* and the US Environmental Protection Agency (EPA) *(2)*. These methods included "smoke" measurements, which may represent the darkness of stain obtained on a white filter-paper through which air has been passed (according to the British smoke method, sometimes referred to as the black smoke method), and also total suspended particulate measurements (gravimetric measurement of particulates of all sizes collected on a glass fibre filter by a high volume sampler according to the method of the US Department of Health, Education, and Welfare *(3)*, as well as by several other methods).

Respirable particles *(1)*, typically with a 4.5 μm aerodynamic diameter (50% cut-off point), are collected by the black smoke method and its variations; some particles up to 7–9 μm are also collected.

Methods to measure total suspended particulates (by high volume sampler) have been used extensively in the USA. There are problems with this method, however, in that the size range of particles sampled extends well beyond those particles that are able to penetrate the upper respiratory tract, and in arid regions the method is liable to sample wind-entrained dust of noncombustive origin. This problem has been recognized by US EPA who recommended that particulate matter of less than 10 μm aerodynamic diameter (PM_{10}) be measured, as a better indicator of health-related particles.

Recommendations have been made by the International Organization for Standardization (ISO) regarding the aerodynamic particle size range corresponding with thoracic penetration *(4)*, and samplers that have acceptance characteristics that approximate that curve are being increasingly used. Such thoracic particle measurements according to the ISO standard (ISO-TP) are roughly equivalent to the sampling characteristics for particulate matter with a 50% cut-off point at 10 μm diameter.

Sources

Sulfur dioxide

There are some natural sources of sulfur dioxide (such as volcanoes) ... bute to environmental levels in the European Region, man-made

contributions from the combustion of fossil fuels are of prime concern in relation to human exposures. Over the past 10–20 years there has been a tendency towards declining emissions in much of the Region, due to changes in the types or amounts of fuel used. More importantly, however, the types of sources have changed even more, away from small multiple sources (domestic, commercial or industrial) towards large single sources such as power stations, which disperse pollutants at higher altitudes. The net result has been a marked reduction in concentrations of sulfur dioxide in many large cities that were at one time highly polluted. A more widespread distribution, by long-distance transport within the Region, is now the dominant pattern.

Acid aerosol

The major proportion of sulfur emissions from combustion sources is emitted as sulfur dioxide, which is further oxidized to sulfur trioxide in the atmosphere at a rate of 0.5–10% per hour. As a result of the presence of moisture, sulfuric acid is formed; this is present as an aerosol, often associated with other pollutants in droplets or solid particles extending over a wide range of sizes. Most of the sulfuric acid in ambient air results from sulfur dioxide emitted by combustion. Other direct or primary point sources of sulfuric acid include acid manufacturing plants and consuming industries, such as fertilizer and pigment factories.

Sulfuric acid and its partial atmospheric neutralization product, ammonium bisulfate, represent almost all of the strong acid content in the ambient aerosol. The ultimate neutralization product, ammonium sulfate ($(NH_4)_2SO_4$), is only weakly acidic. Other strong acids in the ambient air, e.g. nitric acid (HNO_3) and hydrochloric acid (HCl), will be present as vapours, except when incorporated into fog droplets.

Because of its hygroscopic property, sulfuric acid in ambient air will always be present as a solution droplet whose H^+ concentration varies with ambient humidity. Pure ammonium bisulfate can be present as a salt crystal at humidities up to 80%. However, once it is dissolved into droplet form it will not become a crystal again until the humidity falls below 69%. Once inhaled into the moist respiratory tract, it will take up water vapour and deposit as dilute droplets.

Particulate matter

Suspended particulate matter is a term used to cover a range of finely divided solids or liquids that originate from a number of natural or man-made sources.

Particulate matter of respirable size may be emitted from a number of sources, some of them natural (e.g. volcanoes and dust storms) and many others that are more widespread and more important (e.g. power plants and industrial processes, vehicular traffic, domestic coal burning, industrial incinerators). The majority of these non-natural sources are concentrated in limited portions of the territory, i.e. the urbanized areas, where populations are also concentrated *(1,5)*.

Occurrence in air

Sulfur dioxide

As a result of the changes in sources, annual mean levels of sulfur dioxide in major cities of Europe, stated earlier by WHO *(1)* to be within the range 100–200 $\mu g/m^3$, are now largely below 100 $\mu g/m^3$. Similarly, there has been a decline in maximum daily mean values, which are now mainly in the range 250–500 $\mu g/m^3$. Peaks over shorter averaging periods, such as 1 hour, extend to 1000–2000 $\mu g/m^3$ and in some situations higher transient peaks may also occur. Indoor concentrations of sulfur dioxide are generally lower than outdoor concentrations, since absorption of sulfur dioxide occurs on walls, furniture, clothes and in ventilation systems. An exception is occupational exposure, where concentrations of several thousand micrograms may occur regularly *(1)*.

Data on European concentrations of sulfur dioxide and deposition of other sulfur compounds are based either on national monitoring networks, which are largely concentrated in urban areas, or on cooperative programmes for the study of the long-range transport of pollutants *(6,7)*. Natural concentrations of sulfur dioxide are normally below 5 $\mu g/m^3$. The annual mean sulfur dioxide concentrations in most rural areas of Europe are between 5 $\mu g/m^3$ and 25 $\mu g/m^3$. However, as a result of the common practice of using high chimneys to disperse emissions, there are also large rural areas in Europe where average concentrations now exceed 25 $\mu g/m^3$. Sulfur dioxide is often accompanied by elevated levels of nitrogen oxides (NO_x) *(8)*.

Acid aerosol

Current average acid aerosol levels in Europe and North America are not known. The highest current levels reported in recent years have been summarized by Lioy & Lippmann *(9)*. They are in the range of 20–30 μg sulfuric acid per m^3 (6–12 hours average) in various parts of North America, and 28 μg sulfuric acid per m^3 in Europe (Berlin (West)). The highest reported level in the United Kingdom was 680 μg sulfuric acid per m^3 (1-hour average) in London in 1962. Higher levels were almost certainly present in London in earlier years. Maximum ambient concentrations are likely to occur in urban fogs or downwind of coal- and oil-fired power plants and industrial sources. The distribution of secondary acidic aerosol is much more general, ambient levels depending on the rates of sulfur dioxide oxidation and the subsequent neutralization of sulfuric acid in the ambient air by ammonia (NH_3). Rates of sulfur dioxide oxidation depend on ambient temperature, humidity, and concentrations of oxidants and catalytic components of particles in the atmosphere and cloud droplets. Rates of ammonia neutralization depend on the strength of ammonia sources and atmospheric mixing. Ammonia emissions are lowest over water and afforested regions, and higher over urban and agricultural regions. Indoor sources of sulfuric acid are generally not significant except in some occupational environments.

Particulate matter
In rural areas within Europe, black smoke values range from near zero to about 10$\mu g/m^3$. In the larger cities, annual mean concentrations of smoke range from 10 to 40$\mu g/m^3$. Where gravimetric measurements of particulates are made, the annual values lie between about 50 and 150$\mu g/m^3$. Corresponding maxima are 100–250$\mu g/m^3$ (black smoke) and 200–400$\mu g/m^3$ (suspended particulate matter gravimetric).

Conversion factors

Sulfur dioxide

1 ppm = 2860$\mu g/m^3$
1 mg/m^3 = 0.35 ppm

Acid aerosol
Acid aerosol concentrations can be expressed as μmols of H^+/m^3 or as sulfuric acid equivalent in $\mu g/m^3$. There are 98μg per μmol.

Particulate matter
As indicated, no generally applicable conversion factors can be set between black smoke values and various gravimetric particulate matter values (e.g. total suspended particulates or ISO-TP).

Routes of Exposure

Inhalation is the only route of exposure that is of interest in relation to the effects of sulfur dioxide, acidic aerosol and suspended particulate matter on human health. For some special substances, which are constituents such as lead and some highly toxic organic compounds, other routes of uptake such as the alimentary tract may also be of interest. In this context, however, only health effects on the respiratory tract will be considered.

Kinetics and Metabolism

Sulfur dioxide
Absorption of sulfur dioxide in the mucous membranes of the nose and upper respiratory tract occurs as a result of its solubility in aqueous media. The absorption is concentration-dependent, with 85% absorption in the nose at 4–6 mg/m^3 and about 99% at 46 mg/m^3. Only minimal amounts reach the lower respiratory tract *(2,10,11)*. From the respiratory tract, sulfur dioxide enters the blood. Elimination occurs (after biotransformation to sulfate in the liver), mainly by the urinary route.

Acid aerosol
The deposition pattern within the respiratory tract is dependent on the size distribution of the ambient droplets and humidity. Acidic ambient aerosol typically has a mass median aerodynamic diameter of 0.3–0.6μm. Thus,

even with hygroscopic growth in diameter in the respiratory airways by a factor of between 2 and 4, particles remain within the fine-particle range and deposit preferentially in the distal lung airways and airspaces. Some neutralization of the droplets can occur before deposition, due to the normal excretion of endogenous ammonia into the airways. Deposited free H^+ reacts with components of the mucus of the respiratory tract, changing its viscosity *(12)*. The unreacted part of H^+ diffuses into surrounding tissues. The capacity of the mucus to react with H^+ is dependent on the H^+ absorption capacity, which is reduced in acidic saturated mucus as found, for example, in asthmatics.

Under fog conditions the ambient acid is incorporated into droplets, with average droplet sizes in the range of 10–15μm. Such droplets can also contain dissolved nitric acid and other acidic vapours. Inhaled fog droplets will deposit primarily in the upper respiratory tract; very little will penetrate to the deeper lung airways, where most of the fine acidic aerosol will deposit.

Particulate matter
As discussed elsewhere *(1,11,13,14)*, a portion of the inhaled aerosol is deposited by contact with airway surfaces and the remainder is exhaled. In inhalation toxicology, the term "deposition" refers to removal from inspired air of inhaled particles. "Clearance" refers to the subsequent removal of deposited material from the respiratory tract. Within a species, deposition of inhaled particles in the respiratory tract depends mainly on breathing pattern and particle size (aerodynamic diameter). Larger particles (10μm and above) are mainly deposited in the extrathoracic part of the respiratory tract (above the epiglottis) and the main proportion of particles 5–10μm in size are deposited in proximity to the fine airways (respiratory bronchioles) with normal nasal breathing. With mouth breathing, the regional deposition pattern changes markedly, extrathoracic deposition being reduced and tracheobronchial and pulmonary deposition enhanced. The proportion of mouth breathing to nose breathing increases with exercise and conversation *(15)*.

During mouth breathing, fine particles (< 2.5μm aerodynamic equivalent diameter (D_{ae})) deposit primarily in the pulmonary region, between about 3 and 5μm D_{ae} significant deposition in both the pulmonary and the tracheobronchial regions occurs; at larger sizes (about 7–15μm D_{ae}), deposition is predominantly in the tracheobronchial region as opposed to the pulmonary region *(16)*.

Health Effects

Sulfur dioxide

Acute effects
High concentrations of sulfur dioxide can give rise to severe effects in the form of bronchoconstriction and chemical bronchitis and tracheitis, as seen

in animal experiments *(1)* and in occupational exposures to more than 10 000 $\mu g/m^3$. Concentrations of sulfur dioxide in the range 2600–2700 $\mu g/m^3$ give rise to frank effects with bronchospasm in asthmatics *(17)*.

The effects of concern in relation to short-term exposures are those on the respiratory tract. There is an extremely large variability of sensitivity to sulfur dioxide exposure among individuals. This is true for normal persons, but especially so if asthmatics are included *(12)*. Asthmatics have very labile airways and resistance is likely to change in response to many other stimuli, including pollens *(1,2,11)*. Effects observed in asthmatics at relatively low concentrations of sulfur dioxide under laboratory exposure situations are listed in Table 1

Effects of repeated and/or long-term exposures

Repeated short-term occupational exposure to high concentrations of sulfur dioxide combined with long-term exposure to lower concentrations can lead to an increased prevalence of chronic bronchitis, especially in cigarette smokers. A possible contribution of simultaneously occurring sulfuric acid aerosol has, however, not been examined in these studies *(24)*. Several epidemiological studies have associated the occurrence of pulmonary effects in communities with combined exposure to sulfur dioxide and particulates.

A continuum of response to sulfur dioxide exposures at relatively low concentrations has been observed in laboratory investigations of human volunteers. The magnitude of the effects was much enhanced when subjects increased their breathing rates through exercise. The findings in a wide range of studies among asthmatics (Table 1) are consistent with a linear relationship *(25)* between magnitude of effect (in terms of proportionate increase in airway resistance) and dose of sulfur dioxide delivered to the airways (after allowing for removal of a substantial proportion in the nose or mouth). Thus, in a strict sense it would be difficult to define a lowest-adverse-effect level since the effect appears to be a function of the sensitivity of the subject, concentration, duration of exposure (10 minutes being the most usual duration of test exposure), level of activity and mucus rheological properties. It was, nevertheless, considered that effects of concern to the health of exercising asthmatic subjects were demonstrable down to sulfur dioxide levels of about 1000 $\mu g/m^3$, with discernible effects of less certain consequence below that level.

Another aspect, of greater importance to public health, is the proportion of the population liable to be affected. Detailed information regarding the proportion of asthmatic or otherwise sensitive people in the community is not available, although estimates of around 5% have been suggested.

Sensory effects

At concentrations of 10 000 $\mu g/m^3$, sulfur dioxide has a pungent, irritating odour. Since the odour threshold of sulfur dioxide is several thousand $\mu g/m^3$, this criterion is not critical in relation to public health.

Acid aerosol: effects on experimental animals

Acute exposures

Respiratory mechanical function. Alterations of pulmonary function, particularly increases in pulmonary flow resistance, occur after acute exposure. Reports of the irritant potency of various sulfate species are variable *(2,11)*, owing in part to differences in animal species and strains, and also to differences in particle size, pH, composition and solubility. Sulfuric acid is more potent than any of the sulfate salts in terms of increased airway irritancy. For short-term (1-hour) exposures, the lowest concentration of sulfuric acid reported to increase airway resistance was 100 $\mu g/m^3$ (in guinea pigs). The irritant potency of sulfuric acid depends in part on particle size, with smaller particles having more effect.

Particle clearance function. Donkeys exposed by inhalation for 1 hour to 0.3–0.6 μm sulfuric acid at concentrations ranging from 100 to 1000 $\mu g/m^3$ exhibited slowed bronchial mucociliary clearance function at concentrations of $\geq$ 200 $\mu g/m^3$, while rabbits undergoing similar exposures showed an acceleration of clearance at concentrations between 100 and 300 $\mu g/m^3$, and a progressive slowing of clearance at concentrations of $\geq$ 500 $\mu g/m^3$ *(26)*.

Subchronic exposures

Particle clearance function. Donkeys exposed for 1 hour per day, 5 days per week, for 6 months to an aerosol (0.3–0.6 μm) of sulfuric acid at a concentration of 100 $\mu g/m^3$ developed highly variable clearance rates, and a persistent shift from baseline rate of bronchial mucociliary clearance during the exposures and for 3 months after the final exposure. During the 3 months of follow-up, 2 animals had much slower clearance than the baseline rate, while 2 had rates faster than the baseline *(26)*. Rabbits exposed for 1 hour per day, 5 days per week for 20 days to 0.3 μm sulfuric acid at 250 $\mu g/m^3$ developed variable mucociliary clearance rates during the exposure period, and their clearance during a 2-week period following the exposure was substantially faster than their baseline rates *(26)*.

Histology. In the study cited above, in which rabbits were exposed to 250 $\mu g/m^3$ for 4 weeks and sacrificed 2 weeks later, histological examinations of the airways showed increased numbers of secretory cells in distal airways, and thickened epithelial cell layers in airways extending from medium-sized airways to terminal bronchioles. There were no corresponding changes in the trachea or other large airways *(26)*. In a study in which dogs were exposed daily for 5 years to 1100 μg sulfur dioxide per m^3 plus 90 μg sulfuric acid per m^3 and were then allowed to remain in unpolluted air for 2 years, there were small changes in pulmonary functions during the exposure, which continued following the termination of exposure. Morphometric lung measurements made at the end of the two-year post-exposure period showed changes analogous to an incipient stage of human centrilobular emphysema *(14)*.

Table 1. Effects observed in asthmatic subjects during laboratory conditions of exposure to sulfur dioxide

Sulfur dioxide concentration[a] (ppm)	Duration of exposure (min)	Number and type of subject	Type of exposure	Type of activity	Effects[b]	Reference
1, 3, 5	10	7, normal 7, atopic 7, asthmatic	Mouthpiece	Rest	*SRaw* increased significantly at all concentrations for asthmatic subjects, only at 5 ppm for normal and atopic subjects. Some asthmatics exhibited marked dyspnoea requiring bronchodilation therapy	*(18)*
1.0	5	6, asthmatic	Mouthpiece	Exercise	*SRaw* significantly increased in the asthmatic group at 0.5 and 0.25 ppm of sulfur dioxide and at 0.1 ppm in the two most responsive subjects. At 0.5 ppm three asthmatic subjects developed wheezing and shortness of breath	*(19,20)*
0.1, 0.25, 0.5	10	7, asthmatic				
0.50	180	40, asthmatic	Oral chamber Nose clips	Rest	*MMFR* significantly decreased 2.7%; recovery within 30 minutes	*(21)*
0.5	10	5, asthmatic	Mouthpiece	Exercise	*SRaw* increases were observed over exercise baseline rates for 80% of the subjects	*(22)*
0.25, 0.5	60	24, asthmatic	Chamber	Exercise	No statistically significant changes in *FVC* or *SRaw*	*(22)*
0.30	120	19, asthmatic	Chamber	Exercise	No pulmonary effects seen with 0.3 ppm of sulfur dioxide and 0.5 ppm of nitrogen dioxide exposure compared to exercise baseline	(23)

[a] 0.1 ppm of sulfur dioxide = 262 μg/m³; 0.5 ppm = 1310 μg/m³; 1.0 ppm = 2620 μg/m³; 5.0 ppm = 13 100 μg/m³; 10 ppm = 26 200 μg/m³; 50 ppm = 131 000 μg/m³.

[b] Significant increase or decrease noted here refers to "statistically significant" effects, independent of whether the observed effects are "medically significant" or not. Abbreviations are as follows: *SRaw*, specific airway resistance; *MMFR*, maximum mid-expiratory flow rate; *FVC*, forced vital capacity.

Acid aerosol: effects on humans

Acute effects

Respiratory mechanical function. Sulfuric acid and other sulfates have been found to affect both the sensory and the respiratory function in humans.

Respiratory effects from exposure to sulfuric acid (350–500 $\mu g/m^3$) have been reported to include increased respiratory rate and decreased maximal inspiratory and expiratory flow rates and tidal volume *(2,11)*. However, other studies of pulmonary function in nonsensitive healthy adult subjects indicated that pulmonary mechanical function was little affected when subjects were exposed to 100–1000 μg sulfuric acid per m^3 for 10–120 minutes. In one study, the bronchoconstrictive action of carbachol was potentiated by sulfuric acid and other sulfate aerosols, more or less in relation to their acidity. Asthmatics are substantially more sensitive in terms of changes in pulmonary mechanics than healthy people, and vigorous exercise potentiates the effects at a given concentration. The lowest-demonstrated-effect level for sulfuric acid was 100 $\mu g/m^3$ via mouthpiece inhalation in exercising adolescent asthmatics. The effects were relatively small and disappeared within about 15 minutes. In adult asthmatics undergoing similar protocols, the lowest-observed-effect level was 350 $\mu g/m^3$ *(11,27)*.

Particle clearance function. In healthy nonsmoking adult volunteers exposed to 0.5 μm sulfuric acid at rest at 100 $\mu g/m^3$ for 1 hour, there was an acceleration of bronchial mucociliary clearance of particles which deposited primarily in large thoracic airways, and a slowing of clearance when the exposure was raised to 1000 $\mu g/m^3$. For particles that deposited primarily in medium-sized and small airways, there was a small but significant slowing of clearance at 100 $\mu g/m^3$ and a greater slowing at 1000 $\mu g/m^3$. These changes are consistent with the greater deposition of acid in medium-sized to smaller airways. Exposures to 100 $\mu g/m^3$ for 2 hours produced slower clearance than the same exposure for 1 hour, indicating a cumulative relationship to dose *(26)*.

Effects of longer-term exposure

Kitagawa *(28)* identified sulfuric acid as the probable causal agent for approximately 600 cases of respiratory disease in the Yokkaichi area of central Japan between 1960 and 1969. The patients' homes were concentrated within 5 km of a titanium dioxide plant with a 14 m stack that emitted from 100 000 to 300 000 kg sulfuric acid per month in the period 1961–1967. The average concentration of sulfur trioxide in February 1965 in Isozu, a village 1–2 km from the plant, was 130 $\mu g/m^3$, equivalent to a sulfuric acid concentration of 159 $\mu g/m^3$. Kitagawa estimated that peak concentrations might be up to 100 times as high when a north wind was blowing. Electrostatic precipitators were installed to control aerosol emissions in 1967, and after 1968 the number of newly found patients with "allergic asthmatic bronchitis" or "Yokkaichi asthma" gradually decreased. Kitagawa's quanti- [illegible] sulfuric acid and the criteria used to describe cases of respiratory disease may differ from current methods. The unique aspect of this report is the identification of sulfuric acid as the likely causal agent for excess morbidity.

Other evidence of links between high concentrations of ambient sulfuric acid and effects on human health is more circumstantial. Sulfuric acid concentrations in the ambient air were certainly much higher than current levels during the classic episodes in London, the Meuse valley, and Donora, but so were concentrations of many other pollutants. Similarly, the decline in the prevalence of chronic bronchitis in the United Kingdom over [illegible] three decades could have been due to the decline in emissions of any o[illegible] pollutants. However, on mechanistic grounds and in view of known exposure-response relationships, sulfuric acid is a more plausible candidate than sulfur dioxide, carbonaceous particles and other known constituents *(29)*.

In an analysis of 1980 cross-s[illegible]al mortality for the USA *(30)*, predictors of mortality due to air po[illegible]tion were expressed in terms of four aerosol pollutant surrogates, i.e. total suspended particulates, inhalable particles $< 15 \mu m$, fine particles $< 2.5 \mu m$, and sulfate (SO_4^{2-}). Among these, only fine particles and sulfate had statistical significance as predictors of response, but these two surrogates' P values were typically < 0.01.

The measured sulfate includes strong acids (sulfuric acid), the less acidic salt (ammonium bisulfate) and the fully neutralized salt (ammonium sulfate). Since the H^+/SO_4^{2-} ratio is highly variable in time and location and is often close to zero, sulfate is a relatively poor surrogate for acid aerosol concentration. The conclusion that sulfate is a better surrogate for the active component of fine particles than the other three surrogates does not necessarily make it a good one *(29)*. It does, however, lend support to the hypothesis that H^+ is the active agent *(12)*. Unfortunately, epidemiological studies are not available by which mortality and/or morbidity can be related to the acidity (i.e. H^+ ion concentration) of respirable particles *(29)*. This would be expected to constitute a more appropriate measurement *(12)*.

Sensory effects

The odour threshold for sulfuric acid has been estimated to be 750 $\mu g/m^3$ on the basis of one study and 3000 $\mu g/m^3$ on the basis of another *(2)*.

Sulfur dioxide and particulate matter

Short-term health effects related to 24-hour average values of sulfur dioxide and particulate matter

Variations in 24-hour average concentrations of sulfur dioxide, black smoke and total suspended particulates have been associated with increased mortality, morbidity and deficits in pulmonary function tests *(1,2,11)*. Regression analysis of daily pollution variables in relation to urban death rates results in significant coefficients, even after accounting for temperature and other associations. These relationships cannot clearly establish a threshold effect. However, on the basis of the London studies *(31)* in which 24-hour concentrations of sulfur dioxide and black smoke were above 500 $\mu g/m^3$, the daily mortality increased significantly above baseline rates. This does not

preclude the possibility that mortality effects occur below these concentrations. In fact, recent time-series analyses of New York City mortality data over 15 years *(32)* suggest that variations in fine particle measures can explain approximately 5% of the fluctuation in mortality, regardless of weather effects. Concentrations in a range below 500μg black smoke per m^3 were reported in the London analysis, but a different measurement method was used in the report from the USA. Short-term effects of air pollution have been investigated in several studies involving responses in "sensitive" populations. Panel studies of asthmatic individuals have been the most frequently used design *(11)*. Some of the earlier studies, using the responses of asthmatics to varying daily pollution levels, have not been relied upon, primarily because of their small sample size and inadequate exposure measurements. In addition, incidences of illness within a population of bronchitic patients have been studied with respect to daily air pollution concentrations. Significant changes in patients' conditions were observed when black smoke exceeded 250μg/m^3 and sulfur dioxide exceeded 500μg/m^3 *(33)*. Taking into account indications from some other studies, as in the earlier WHO report *(1)*, the minimum level of smoke and sulfur dioxide needed to produce effects was taken as 250μg/m^3.

In some studies, deviations in pulmonary function measures have been observed in children and adults that are associated with short-term fluctuations in particulate concentrations *(1,2,11,34,35)*. In another study of approximately 1000 children living in an industrialized community, a statistically significant negative mean slope of forced vital capacity (FVC) and forced expiratory volume (FEV) was found for total suspended particulates (11–272μg/m^3) and sulfur dioxide (0–281μg/m^3), with a correlation coefficient $r = 0.75$ *(36)*. In this study total suspended particulate measurements were complemented by parallel inhalable particle measurements *(37,38)*. Since inhalable particle values are generally similar to thoracic particle values, it was possible to estimate total suspended particulates/ISO-TP ratios. From the data collected by Dockery et al. *(36)* it can be calculated that in those 25% of children who were most sensitive, there was at least a four times greater deficit in pulmonary function compared with those of average sensitivity (for this subgroup a decrease in FEV of 0.39 ml/μg per m^3 was observed). Those effects are associated with concentrations of total suspended particulates in the range of 150–200μg/m^3 (in the presence of sulfur dioxide), although total suspended particulate concentrations have frequently exceeded 260μg/m^3. Minimum levels for effects were judged to be 180μg/m^3 in the presence of sulfur dioxide. Relating total suspended particulates to ISO-TP would result in the same deficit in pulmonary function at concentrations of thoracic particles above 110μg/m^3 in the presence of sulfur dioxide. These values are estimated using specific total suspended particulates/ISO-TP ratios *(37)*.

Although these changes are of health concern, the physiological significance of such apparently reversible effects on the immediate or long-term health of the individual is unknown.

In Table 2 the evidence on short-term health effects is summarized in terms of the lowest-observed-effect levels of air pollutants on health.

Table 2. Summary of effects on human health of lowest-observed-effect levels of sulfur dioxide and particulate matter (short-term exposure)

Effect	24-hour mean exposure to:			
	SO_2 (μg/m^3)	smoke (μg/m^3)	total suspended particulates (μg/m^3)	thoracic particles (μg/m^3)
Excess mortality	500	500		
Increased acute respiratory morbidity (adults)	250	250		
Decrements in lung function (children)			180	110

Long-term health effects related to annual means of sulfur dioxide and particulate matter

Mortality. Variations in mortality (all causes) and, more specifically, in mortality from cardiorespiratory diseases have been found during comparison of the findings from different cities in several countries *(1)*. Multiple-regression analyses, using various indices of pollution (as long-term means), together with socioeconomic factors, indicate associations with pollutants (particulates and sulfate being the ones generally incorporated in analyses in the USA) that account for a small proportion (about 4%) of the variation in death rates between cities *(30,39–41)*. Thus, it could be said that there are discernible effects of long-term exposure to the pollution complex of the particulate matter/sulfur dioxide type at relatively low annual mean levels, but it is considered that no firm guidance on lowest-observed-effect levels can be given on the basis of relationships of this type.

Morbidity. Further epidemiological studies on differences in the prevalence of respiratory symptoms (adults and children) and the frequency of respiratory illness (children) between communities with differing levels of pollution have provided results that are consistent with the conclusions reached earlier by WHO *(1)*, indicating detectable increases where annual mean concentrations of both black smoke and sulfur dioxide exceed 100μg/m^3 *(42,43)*. Other pollutants, such as sulfates (or acid sulfates) may be relevant, but no measurements were available in the studies in question. The more recent studies have mainly been analysed using multiple-regression models, taking confounding variables into account as far as possible *(44,45)*. In this way, the relative importance of different factors

has been shown more clearly and relationships are taken to be continuous, indicating that effects may well extend below the pollution levels quoted.

Community-based health studies are useful in attributing excess illness rates or differences in pulmonary performance to air pollution. Communities differ for a variety of cultural, social, economic and other factors that can result in different frequencies of illness. While air pollution may contribute to elevated illness rates, it is difficult to describe with certainty a level, an averaging time or even a specific contaminant that is unequivocally associated with a threshold effect level. Increased age-adjusted illness rates are associated with indices for sulfur dioxide, black smoke, total suspended particulates, and fine particles in several studies *(1,2,11)*. Community differences in illness rates can be discerned in several more contemporary studies conducted in the late 1970s and early 1980s. It is of interest to note that the annual sulfur dioxide and total suspended particulate concentrations are lower than the concentrations associated with effects in earlier studies. For instance, in the USA *(46)* differences in community illness rates have been associated with annualized total suspended particulate concentrations ranging from 30 to 100 $\mu g/m^3$ (20–55 $\mu g/m^3$ when the particles measure less than 10 μm in diameter). The two communities with the highest illness rates had particle concentrations (for particles less than 10 μm in diameter) of 35 and 55 $\mu g/m^3$ (annual means).

In the Netherlands, a decreasing difference in respiratory symptom rates between a polluted and a cleaner area was observed *(47)*. Initially, annual average sulfur dioxide concentrations above 200 $\mu g/m^3$ were observed in the polluted area, but after the mid-1970s sulfur dioxide levels were between 45 and 80 $\mu g/m^3$, while black smoke decreased from 34–45 to 25–35 $\mu g/m^3$. In the cleaner area sulfur dioxide values, measured after 1975, were 10–25 $\mu g/m^3$, and black smoke levels, measured after 1982, were 10–15 $\mu g/m^3$. In France *(48)* differences in symptom rates are associated with annual averages of sulfur dioxide over a range of 13–127 $\mu g/m^3$, measured by acidimetry, or a range of 22–85 $\mu g/m^3$, as measured by a specific technique.

Decrements in lung function. Measurements of respiratory physiology were included in several of the studies referred to above. Several of these observations have been reviewed by WHO *(1)*, EPA *(2)* and Ericsson & Camner *(11)*. Studies that have been conducted in the same communities over a period of years show associations between the magnitude of lung function changes and the levels of pollution. One series of such studies, carried out in the USA *(49–51)*, indicated effects associated with particulates (measured as total suspended particulates) at an annual mean of 180 $\mu g/m^3$, though documentation of pollution levels in the series as a whole was incomplete and other pollutants could have been involved. From a more extensive series carried out in the Netherlands *(47)* it has been concluded that consistently lower lung function values in an urban, as compared with a rural, area might point to long-term effects of pollution. While much current information on a wide range of pollutants was available, it was considered that the effect could have related to earlier higher

levels, and no firm guidance can be given at this stage in relation to lowest-observed-adverse-effect levels.

Sensory effects

Community exposure to urban air pollutants, including sulfur oxides, nitrogen oxides and particulate matter, may give rise to feelings of discomfort, which can only be assessed subjectively by those persons who are affected *(1,52)*. Annoyance reactions to urban air pollutants are common phenomena. In a Swedish study *(52)* of population groups in central Stockholm, 60% of the population reported annoyance of this kind. One quarter of those were classed as being very annoyed. Comparative studies in suburban areas and smaller Swedish towns disclosed lower prevalence figures for annoyance. Surveys of annoyance are fraught with many problems *(1)*. Since annoyance reactions have a large sociocultural component, prevalence figures in relation to air pollution levels may vary from place to place and should be determined for each locality.

Evaluation of Human Health Risks

Sulfur dioxide

When using the evidence from human experimental studies of sulfur dioxide to draw up recommendations for guideline values aimed at protecting people from the risk of adverse effects, the need to avoid brief exposures to peak values is implied. Some protection (safety) factor may have to be incorporated when using information on the lowest-observed-effect level in order to protect especially sensitive asthmatic patients (who have not been subject to testing), though they would be less likely to be involved in exercise at the levels used in the experimental exposures. In relation to a lowest-observed-effect level of concern to health of 1000 $\mu g/m^3$ (10 minutes), it appears reasonable to apply a protection (safety) factor of 2 for the protection of public health; this would give a concentration of 500 $\mu g/m^3$ (10 minutes). The occurrence of such concentrations can often be predicted from the frequency distribution of locally measured concentrations, by using some existing models for averaging values over different time periods in the case of diffuse or multiple sources *(53)*.

Predictions for point sources can also be made if the characteristics of the source and the local diffusion conditions are known *(54)*. Frequency distribution characteristics can also help in guiding authorities towards solutions. These frequency distributions are known for a large number of towns in Europe *(55)* and the USA *(56)*. As an example, if the aim were to ensure that the 10-minute mean value of 500 $\mu g/m^3$ was not exceeded, then on the basis of calculations of multiple-source situations in the Netherlands *(55)*, the corresponding 1-hour value that should not be exceeded would be 350 $\mu g/m^3$

Acid aerosol

While the data currently available are insufficient to establish a numerical guideline, they do raise serious concern that acidic aerosol could account

for past associations between particulate air pollution and the exacerbation and development of chronic bronchitis.

Recent 1-hour acute experimental inhalation exposure data on humans and two animal species (donkeys and rabbits) show similar exposure–response relationships in terms of transient and reversible changes in the rate of tracheobronchial mucociliary clearance. Comparable exposures, when repeated on a daily basis in the two animal species, produced persistent changes in clearance rates, and in the one species in which histological examinations were made, changes in the airways after only 20 days of exposure were of a similar character to those seen in young human smokers examined at autopsy. The analogy with cigarette smoke, which is a known causal factor in chronic bronchitis, has been pointed out by Lippmann *(29)*.

The association shown in Japan (Yokkaichi) between sulfuric acid aerosols and respiratory morbidity gives support to the hypothesis that acid aerosol is an important component of urban air pollution. This hypothesis is also consistent with the results of cross-sectional studies of daily mortality in major cities in the USA, which indicate that sulfate is a better predictor for mortality than any of the nonspecific gravimetric indices that have been used.

More data on human exposures are clearly needed to test the hypothesis of causality. Situations that would be of concern for monitoring purposes would be those where humans were exposed repeatedly to concentrations at or above 10 $\mu g/m^3$ (sulfuric acid or equivalent acidity of aerosol).

Sulfur dioxide and particulate matter

The lowest-observed-effect levels for short-term and long-term (annual mean) average air pollution measurements are summarized in Tables 2 and 3. Evaluation of the measured components of air pollution in relation to public health is, however, difficult for a number of reasons noted in the

Table 3. Summary of effects on human health of lowest-observed-effect levels of sulfur dioxide and particulate matter (long-term exposure)

Effect	Annual mean exposure to:		
	SO_2 ($\mu g/m^3$)	smoke ($\mu g/m^3$)	total suspended particulates ($\mu g/m^3$)
Increased respiratory symptoms or illness	100	100	
Decrements in lung function			180

WHO publication *(1)*. A number of these points still remain largely unresolved. For example, it is not clear whether long-term effects can be related simply to annual mean values or to repeated exposures to peak values. Similarly, it remains uncertain which components of the sulfur dioxide/particulates complex are involved in the adverse effects, though increasingly attention is being given to the role of secondary products such as acid sulfates. Arbitrary protection (safety) factors of 2 in relation to the morbidity and mortality data, and 1.5 for decrements in lung functions (considered to represent a less severe effect), seem to be appropriate according to the present state of knowledge.

Measurements of black smoke can no longer be interpreted in terms of $\mu g/m^3$ in many localities, and decisions have already been made (by ISO) to abandon any attempt at mass equivalence. The method is still of value as an index of soiling capacity and of the type of pollution (coal smoke) that has been associated in the past with adverse health effects, and to provide continuity with any further epidemiological studies. Therefore, observations should be continued.

Various direct gravimetric measurements have been used in recent decades, notably the total suspended particulate measurements (by high volume sampler) in the USA. There are problems, however, with the wide size range of particles sampled and the influence of wind-entrained dust. Although a large body of data on such measurements exists, it is now considered misleading to attempt to specify guidelines in terms of total suspended particulates.

Total suspended particulate measurements may, nevertheless, be used for comparison with newer indices of pollution, and they may be of value as a supplement to gravimetric ISO-TP measurements, especially in areas where there is special concern about larger particles.

Efforts should now be made to establish a method of gravimetric measurement representing more realistically the size range of particles that can be inhaled into the thoracic region, even though uncertainties must remain about the component or components most relevant to health. Recommendations have already been made by ISO regarding the (aerodynamic) particle size range corresponding with thoracic penetration, and it is proposed that samplers should have acceptance characteristics that approximate to that curve.

The inclusion of the somewhat wider size range of particles than those sampled by the black smoke method would mean that, even in areas where coal smoke still forms a dominant part of the suspended particulates, results from these gravimetric instruments would be somewhat higher than might be obtained from co-located smoke samplers. Thus, in those circumstances the corresponding guidelines would be a little higher in true gravimetric terms (possibly by about 10%). Now that the characteristics of present-day pollution differ from those of coal smoke pollution, the old data cannot be used with any confidence as a basis for guidelines.

In view of the considerable uncertainties involved in formulating guidelines for particulate matter, there is a need for further epidemiological studies, particularly in those areas where high concentrations will occur.

using well defined methods for particulate measurement and epidemiological assessment, including the control of possible confounding factors such as smoking.

Guidelines

Sulfur dioxide

It appears reasonable to apply a protection factor of 2 for the protection of public health; a guideline value of 500 $\mu g/m^3$ (10 minutes, not to be exceeded) is recommended. A 1-hour maximum value that conforms with this guideline can be calculated as approximately 350 $\mu g/m^3$.

Acid aerosol

Recommendations for air quality guideline values for the strong acid content of ambient aerosol cannot now be made owing to the sparsity of current data on effects and ambient exposure levels. However, monitoring is warranted when levels (sulfuric acid or equivalent acidity of aerosol) exceed 10 $\mu g/m^3$. Therefore, ambient air should be regularly monitored for the H^+ ion concentration of the aerosol (which should be sampled in a size-fractionating particulate sampler) when levels of this magnitude are likely to occur.

Combined effects

In proposing guidelines based on the present knowledge of exposure to both sulfur dioxide and particulate matter, an arbitrary protection (safety) factor of 2 has been used in relation to morbidity and mortality, and a factor of 1.5 has been used for the decrement in lung function, which is considered to be a less severe effect. The recommended guideline values are shown in Table 4.

References

1. *Sulfur oxides and suspended particulate matter.* Geneva, World Health Organization, 1979 (Environmental Health Criteria, No. 8).
2. *Air quality criteria for particulate matter and sulfur oxides,* Vol. I, II & III. Research Triangle Park, NC, US Environmental Protection Agency, 1982 (EPA-600/8-82-029a, b & c).
3. *Air pollution measurements of the National Air Sampling Network, 1957–1961.* Cincinnati, OH, US Department of Health, Education, and Welfare, 1962, pp. 3–4.
4. *Air quality — particle size fraction definitions for health-related sampling.* Geneva, International Organization for Standardization, 1983 (Technical Report ISO/TR 7708-1983 (E)).
5. Proceedings of the Symposium on Biological Tests in the Evaluation of Mutagenicity and Carcinogenicity of Air Pollutants with Special Reference to Motor Exhausts and Coal Combustion Products [illegible] *Environmental health perspectives,* 47: 1–324 (1983).
6. *Manual for sampling and chemical analysis.* Lillestrøm, Norwegian Institute for Air Research, 1977 (EMEP/CHEM 3/77).

Table 4. Guideline values for combined exposure to sulfur dioxide and particulate matter[a]

Averaging time	Sulfur dioxide ($\mu g/m^3$)	Gravimetry: Reflectance assessment, black smoke[b] ($\mu g/m^3$)	Gravimetry: Total suspended particulates (TSP)[c] ($\mu g/m^3$)	Gravimetry: Thoracic particles (TP)[d] ($\mu g/m^3$)
Short term	24 hours	125	120[e]	70[e]
Long term	1 year	50	—	—

[a] No direct comparisons can be made between values for particulate matter in the right- and left-hand sections of this table, since both the health indicators and the measurement methods differ. While numerically TSP/TP values are generally greater than those of black smoke, there is no consistent relationship between them, the ratio of one to the other varying widely from time to time and place to place, depending on the nature of the source.

[b] Nominal $\mu g/m^3$ units assessed by reflectance. Application of the black smoke values is recommended only in areas where coal smoke is the predominant component of the particulates. It does not necessarily apply where diesel smoke is an important contributor.

[c] TSP measurement by high volume sampler without any size selection.

[d] TP equivalent values as for a sampler with ISO-TP characteristics (having 50% cut-off point at 10 μm), estimated from TSP values using site-specific TSP/ISO-TP ratios.

[e] Values to be regarded as tentative at this stage, being based on a single study involving sulfur dioxide exposure also.

7. *International operations handbook for measurement of background atmospheric pollution.* Geneva, World Meteorological Organization, 1978.
8. Fowler, D. & Cape, J.N. Air pollutants in agriculture and horticulture. *In:* Unsworth, M.H. & Ormrod, D.P., ed. *Effects of gaseous air pollution in agriculture and horticulture.* London, Butterworth, 1982, pp. 3–26.
9. Lioy, P.J. & Lippmann, M. Measurement of exposure to acidic sulfur aerosols. *In:* Lee, S.D. et al., ed. *Aerosols.* Chelsea, MI, Lewis, 1986.
10. *Air quality in selected urban areas, 1973–1974.* Geneva, World Health Organization, 1976 (WHO Offset Publication No. 30).
11. Eriksson, G. & Camner, P. Health effects of sulfur oxides and particulate matter in ambient air. *Scandinavian journal of work, environment & health,* 9(Suppl. 3): 1–52 (1983).
12. Holma, B. Influence of buffer capacity and pH-dependent rheological properties of respiratory mucus on health effects due to acidic pollution. *Science of the total environment,* 41: 101–123 (1985).
13. Lippmann, M. et al. Deposition, retention, and clearance of inhaled particles. *British journal of industrial medicine,* 37: 337–362 (1980).
14. Stara, J.F. et al. *Long-term effects of air pollutants in canine species.* Research Triangle Park, NC, US Environmental Protection Agency, 1980 (Report No. EPA-600/8-80-014).
15. Camner, P. & Bakke, B. Nose or mouth breathing? *Environmental research,* 21: 394–398 (1980).
16. *Second addendum to air quality criteria for particulate matter and sulfur oxides.* Washington, DC, US Environmental Protection Agency, 1986 (EPA-600/8-86-020A).
17. Islam, M.S. & Ulmer, W.T. Untersuchungen zur Schwellenkonzentration von Schwefeldioxyd bei besonders Gefährdeten [Borderline concentrations of SO_2 for patients with oversensitivity of the bronchial system]. *Wissenschaft und Umwelt,* 1: 41–47 (1979).
18. Sheppard, D. et al. Exercise increases sulfur dioxide-induced bronchoconstriction in asthmatic subjects. *American review of respiratory disease,* 123: 486–491 (1981).
19. Sheppard, D. et al. Lower threshold and greater bronchomotor responsiveness of asthmatic subjects to sulfur dioxide. *American review of respiratory disease,* 122: 873–878 (1980).
20. Sheppard, D. et al. Inhibition of sulfur dioxide-induced bronchoconstriction by disodium cromoglycate in asthmatic subjects. *American review of respiratory disease,* 124: 257–259 (1981).
21. Jaeger, M.J. et al. Effect of 0.5 ppm sulfur dioxide on the respiratory function of normal and asthmatic subjects. *Lung,* 156: 119–127 (1979).
22. Linn, W.S. et al. Respiratory responses of young adult asthmatics to sulfur dioxide exposure under simulated ambient conditions. *Environmental research,* 29: 220–232 (1982).
23. Linn, W.S. et al. Respiratory effects of mixed nitrogen dioxide and sulfur dioxide in human volunteers under simulated ambient exposure conditions. *Environmental research,* 22: 431–438 (1980).

24. Stjernberg, N. et al. Prevalence of bronchial asthma and chronic bronchitis in a community in Northern Sweden, relation to environmental and occupational exposure to sulphur dioxide. *European journal of respiratory diseases,* 67: 41–49 (1985).
25. Kleinman, M.T. Sulfur dioxide and exercise: relationships between response and absorption in upper airways. *Journal of the Air Pollution Control Association,* 34: 32–37 (1984).
26. Schlesinger, R.B. The effects of inhaled acid aerosols on lung defenses. *In:* Lee S.D. et al., ed. *Aerosols.* Chelsea, MI, Lewis, 1986.
27. Utell, M.J. & Morrow, P.E. Effects of inhaled acid aerosols on human lung function: studies in normal and asthmatic subjects. *In:* Lee, S.D. et al., ed. *Aerosols.* Chelsea, MI, Lewis, 1986.
28. Kitagawa, T. Cause analysis of the Yokkaichi asthma episode in Japan. *Journal of the Air Pollution Control Association,* 34: 743–746 (1984).
29. Lippmann, M. Airborne acidity: estimates of exposure and human health effects. *Environmental health perspectives,* 63: 63–70 (1985).
30. Evans, J.S. et al. Cross-sectional mortality studies and air pollution risk assessment. *Environment international,* 10: 55–83 (1984).
31. Martin, A.E. & Bradley, W.H. Mortality, fog and atmospheric pollution An investigation during the winter of 1958–59. *Monthly bulletin of the Ministry of Health Public Health Laboratory Service,* 19: 56–72 (1960).
32. Schimmel, H. Evidence for possible acute health effects of ambient air pollution from time series analysis: methodology questions and some new results based on New York City daily mortality, 1963–1976. *Bulletin of the New York Academy of Medicine,* 54: 1052–1108 (1978).
33. Lawther, P.J. et al. Air pollution and exacerbations of bronchitis. *Thorax,* 25: 525–539 (1970).
34. Cohen, A.A. et al. Symptom reporting during recent publicized and unpublicized air pollution episodes. *American journal of public health,* 66: 442–449 (1974).
35. van der Lende, R. et al. A temporary decrease in the ventilatory function of an urban population during an acute increase in air pollution. *Bulletin européen de physiopathologie respiratoire,* 11: 31–43 (1975).
36. Dockery, D.W. et al. Change in pulmonary function in children associated with air pollution episodes. *Journal of the Air Pollution Control Association,* 32: 937–942 (1982).
37. Spengler, J.D. et al. *Comparison of hi-vol, dichotomous, and cyclone samplers in four US cities.* 73rd Annual Meeting of the Air Pollution Control Association, Montreal, Quebec. 22–27 June, 1980 (unpublished paper)
38. Spengler, J.D. & Thurston, G.D. Mass and elemental composition of fine and coarse particles in six US cities. *Journal of the Air Pollution Control Association,* 33: 1162–1171 (1983).
39. Lave, L.B. & Seskin, E.P. Air pollution, climate, and home heating: their effects on US mortality rates. *American journal of public health,* 62: 909–916 (1972).
40. Lave, L.B. & Seskin, E.P. *Air pollution and human health.* Baltimore, MD, Johns Hopkins University Press, 1977.

41. Chapple, M. & Lave, L. The health effects of air pollution: a [illegible] analysis. *Journal of urban economics*, 12: 346–376 (1982)
42. Luna, J.E. et al. Patterns of respiratory illness in Sheffield [illegible] schoolchildren. *British journal of preventive and social medicine*, 21: 7–16 (1967)
43. Luna, J.E. et al. Patterns of respiratory illness in Sheffield [illegible] schoolchildren — a follow-up study. *British journal of preventive and social medicine*, 24: 223–228 (1970).
44. Ostro, B. The effects of air pollution and community health. *Journal of the Royal College of Physicians (London)*, 5: 362–368 (1983)
45. Wojtyniak, B. et al. Importance of urban air pollution in chronic respiratory problems. *Zeitschrift für Erkrankungen der Atmungsorgane*, 163: 274–284 (1984).
46. Ware, J.H. et al. Effects of ambient sulfur oxides and suspected particles on respiratory health of preadolescent children. *American review of respiratory disease*, 133: 834–842 (1986).
47. van der Lende, R. et al. Longitudinal epidemiological studies on effects of air pollution in the Netherlands. *In:* Lee, S.D. et al., ed. *Aerosols*. Chelsea, Lewis, 1986
48. PAARC Cooperative Group. Atmospheric pollution and chronic or recurrent respiratory diseases. I. Methods and material. II. Results and discussion. *Bulletin européen de physiopathologie respiratoire*, 18: 87–99, 101–116 (1982).
49. Ferris, B.G., Jr & Anderson, D.O. The prevalence of chronic respiratory disease in a New Hampshire town. *American review of respiratory disease*, 86: 165–185 (1962).
50. Ferris B.G. et al. Chronic non-specific respiratory disease, Berlin, New Hampshire, 1961–67: a cross-section study. *American review of respiratory disease*, 104: 232–244 (1971).
51. Ferris, B.G. et al. Chronic non specific respiratory disease in Berlin, New Hampshire 1967–1973. A further follow-up study. *American review of respiratory disease*, 113: 475–485 (1976)
52. Sörensen, S. et al. Annoyance reactions. *In:* Ewetz, L. & Camner, P., ed. *Health risks resulting from exposure to motor vehicle exhausts*: report to the Swedish Government Committee on Automotive Air Pollution. Stockholm, National Institute of Environmental Medicine, 1983.
53. Larsen, R.I. *A mathematical model for relating air quality measurements to air quality standards*. Research Triangle Park, NC, US Environmental Protection Agency, 1971 (Report No. AP-89).
54. Pasquill, F. Atmospheric dispersion of pollution. *Quarterly journal of the Royal Medical Society*, 97: 369–395 (1971).
55. National Air Pollution Monitoring Network 1984. Bilthoven, National Institute of Public Health and Environmental Hygiene, 1984 (Report Nml-RIUm No. 28).
56. *Health consequences of sulfur oxides: a report from CHESS, 1970–71*. Research Triangle Park, NC, US Environmental Protection Agency, 1974 (Report No. EPA-650/1-74-004).

31

Vanadium

General Description

Vanadium (V) is a bright white ductile metal belonging to group V of the periodic system of elements. It forms compounds mainly in valence states +3, +4 and +5. In the presence of oxygen, air or oxygenated blood, or oxidizing agents, vanadium is always in the +5 oxidation state. In the presence of reducing agents, vanadium compounds are in the +4 oxidation state *(1)*. Vanadium forms both cationic and anionic salts, and can form covalent bonds to yield organometallic compounds which are mostly unstable.

Sources

Vanadium is an ubiquitous metal. The average concentration of vanadium in the earth's crust is 150 µg/g *(2)*. concentrations in soil vary in the range 3–310 µg/g *(3)* and may reach high values (up to 400 µg/g) in areas polluted by fly ash *(4)*. The concentration of vanadium in water is largely dependent on geographical location and ranges from 0.2 to more than 100 µg/litre in freshwater *(2)*, and from 0.2 to 29 µg/litre in seawater *(3)*. The ocean floor is the main long-term sink of vanadium in the global circulation *(4)*. The concentrations of vanadium in coal and crude petroleum oils vary widely (1–1500 mg/kg) *(2)*.

The world production of vanadium was about 35 000 tonnes in 1981 *(5)*, the major producing countries being Chile, Finland, Namibia, Norway, South Africa, USSR and the United States.

Most of the vanadium produced is used in ferrovanadium and, of this, the majority is used in high-speed and other alloy steel (usually combined with chromium, nickel, manganese, boron and tungsten).

It has been estimated that around 65 000 tonnes of vanadium annually enter the environment from natural sources (crustal weathering and volcanic emissions) and around 200 000 tonnes as a result of man's activities *(6)*. The major anthropogenic point sources of atmospheric emission are metallurgical works (3 kg per tonne of vanadium produced), followed by the burning of crude oil, residual oil and coal (0.2–2 kg per 1000 tonnes and [illegible] per 10⁶ litres) *(3)*.