Recommendation from the Scientific Committee on Occupational Exposure Limits for Sulphuric acid

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Employment,
Social Affairs
and Inclusion

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8-hour TWA:	0.05 mg/m$^{3, a}$
STEL (15 min):	(a recommendation of 0.1 mg/m^3 is desirable, but at present a suitable measurement method is not available)
Notation:	none

[a] As the inhalable fraction (see Annex 2, December 2012).

Substance identification

Chemical Name	Sulphuric Acid
CAS No:	7664-93-9
EEC Index No:	016-020-00-8
Formula:	H_2SO_4
Synonyms:	battery acid, hydrogen sulphate, oil of vitriol
Boiling point:	290C
Melting point:	10.36C
Specific gravity	1.84
Vapour pressure:	<0.001 torr at 20C
Conversion factor:	1 ppm = 4.07 mg/m^3 at 20C
Classification	C; R35 Causes severe burns

Sulphuric acid is a dense, oily, colourless and odourless liquid with low vapour pressure. Impurities tend to produce a brown discolouration. It is hygroscopic and soluble in water although the addition of water can produce a violent, exothermic reaction. Because of its hygroscopic nature, particle size of aerosols is influenced by humidity. Concentrated sulphuric acid is corrosive to metals but non-flammable.

1. Occurrence and Use

Sulphuric acid is mainly produced via the catalytic reaction of sulphur dioxide with oxygen to produce sulphur trioxide. This is subsequently dissolved in water to produce concentrated sulphuric acid (over 99% pure). H_2SO_4 is widely used in chemicals manufacture and industrial processes e.g. batteries, detergents, fertilizers, explosives, pharmaceuticals, petroleum, steel, paper products and textiles, and other processes such as metal cleaning and electroplating.

2. Health effects

2.1. Toxicokinetics

Sulphuric acid is highly soluble in water and readily absorbed from the upper respiratory tract following inhalation exposure (DFG, 2001). In the warm, humid conditions of the human upper respiratory tract, there is likely to be an increase in the particle size of inhaled aerosols, because of the hygroscopic nature of H_2SO_4. Larger droplets (10-15 µm) deposit in the nose and smaller droplets (1-10 µm) would reach more deeply within the respiratory tract (larynx, trachea and bronchi). Once absorbed, the sulphate ions formed become indistinguishable from sulphate derived from dietary sources. There are no data describing the extent of dermal absorption of the aerosol or liquid. Its polarity suggests that there would be little significant absorption by this route unless the acidity caused skin damage resulting in the breaching of the skin barrier.

2.2. Acute toxicity

Substantial information is available on the acute effects of exposure to sulphuric acid. A number of human case reports are available in which single inhalation exposure to H_2SO_4 mist has been associated with local eye and respiratory tract irritation, and dental blackening (DFG, 2001). Reactions have been severe (eg. including blood being expectorated). The exposure levels causing such effects have not been adequately described.

A number of volunteer studies are available in which asthmatic and non-asthmatic subjects have been exposed for brief durations to H_2SO_4 aerosols. Although it could be expected that asthmatics would be more sensitive to the effects of H_2SO_4 aerosols on the respiratory tract, the available information does not indicate that this is the case. At exposure levels of 20 mg/m^3 for 30-60 minutes, intense coughing, lacrimation and rhinorrhoea were reported (Sim and Pattle, 1957). At around 2 mg/m^3 for 1 hour with intermittent exercise, slight changes in lung function were noted in asthmatics and non-asthmatics (Avol et al, 1988). At exposure levels of 0.8-1 mg/m^3, there was increased reporting of throat irritation (coughing) amongst asthmatics and non-asthmatics (Frampton et al, 1992). In general, most studies indicated that there were no adverse respiratory tract effects observable in lung function or bronchial reactivity testing on single exposure to around 0.5 mg/m^3 (DFG, 2001). Some studies conducted at exposure levels in the region 0.5-2 mg/m^3 report no adverse findings (DFG, 2001). The differences in findings between different studies may be attributable to inter-individual variation as well as the differing experimental conditions (such as particle size, humidity, intensity of exercise). However, there is inadequate

information to draw conclusions about the relative contribution of each of these variables.

Several studies have examined tracheobronchial clearance with short-term exposure to sulphuric acid mist at concentrations of about 0.1-1.0 mg/m^3 (see DFG, 2001). The results for similar exposure concentrations vary between studies; there were clear, statistically significant reductions in mucociliary clearance at about 1 mg/m^3 , with more inconsistent and generally not statistically significant changes at 0.3 mg/m^3 and below.

The acute toxicity of H_2SO_4 has been studied in a wide range of laboratory animals. The main effects observed are corrosion or irritation of the skin, eyes or upper respiratory tract (DFG, 2001). It is acutely toxic by inhalation of the aerosol mist with rat 1-hour and 2-hour LC_{50} values of around 85-103 mg/m^3 and 510 mg/m^3 respectively. A mouse 8-hour LC_{50} is cited as 500 mg/m^3 and 8-hour LC_{50}s in the range 18-109 mg/m^3 have been reported for the guinea pig. Inconsistencies in the LC_{50} values may be partly due to the influence of humidity and droplet size; there is evidence from guinea pig studies that aerosol droplets with a MMAD around 1µm are more toxic than smaller droplets (eg. 0.4 µm) (Turner and Fairhurst, 1992). There are also likely to be species differences in the response to inhalation exposure. From the species that have been investigated, it would seem that the most sensitive is the guinea pig. This is due to pronounced bronchoconstriction and laryngospasm, a response that occurs in this species upon exposure to irritant gases (Griffiths, 1996). Other rodent species do not display such a severe response.

The effects of a single exposure to sub-lethal concentrations with particle size MMAD 0.3 µm have been extensively studied in rabbits (DFG, 2001). Some slight and variable changes in bronchial parameters were noted in different studies at concentrations in the range 0.075-0.26 mg/m^3, but the significance of the findings in relation to predicting the effects of such exposures on human health is unclear. At around 1 mg/m^3 (the highest exposure level tested) for 1 hour there was slowed mucociliary clearance, mild inflammatory reaction in the lungs, and increased polymorphonuclear counts (in BALF). Studies in guinea pigs exposed at around 1 mg/m^3 for 1-6 hours (MMAD 1 µm) showed slowed mucociliary clearance, thickening of tracheal mucus, and decreased lung compliance/increased airways resistance. At 20 mg/m^3 (MMAD 1 µm) for 4 hours effects on the lung were severe and included dyspnoea, partial atelactasis, loss of ciliated cells in the upper airways, desquamation, oedema, inflammatory infiltration in alveoli, and bronchoconstriction.

2.3. Skin and eye irritation

In relation to the effects of contact with the liquid, concentrated H_2SO_4 is corrosive to the skin and eyes There is no clear quantitative information about skin or eye irritation potential from airborne H_2SO_4 (DFG, 2001).

2.4. Sensitisation

There are no studies of skin sensitisation potential. However, such a response would not be expected as protein binding and hapten formation would seem to be most unlikely. There are no data regarding asthma induced by sulphuric acid although, if it occurred at all, it would be unlikely to result from an immunological mechanism.

2.5. Effects of repeated exposure

There is information available on the repeated exposure effects of H_2SO_4 both in humans and in experimental animals. The data from humans is of limited value, mainly because of historical difficulties in measuring airborne concentrations. One unpublished study notes an increased prevalence of itching eyes, nasal irritation, nasal discharge, sneezing, nose bleeds, throat irritation, dry nose and cough reported in association with occupational exposures which were stated to be 'less than 0.15 mg/m$^{3'}$ (Foster et al, 1996). The prevalence increased amongst workers exposed to higher exposure levels (given as 0.15-0.5 mg/m^3). A further report has been published recently on the already well-documented observation of dental erosion associated with occupational exposure to H_2SO_4 (Chikte and Josie-Perez, 1999). An increased prevalence and severity of dental erosion with increased exposure is reported (96% in workers said to be working in prevailing atmospheric conditions of 0.3-1.0 mg/m^3 H_2SO_4 compared to 75% in workers said to be working in prevailing atmospheric conditions of 0.1-0.3 mg/m^3 H_2SO_4). However, as the authors themselves acknowledge, the reliability of the stated concentration ranges as representative of the personal exposures involved is very doubtful.

Repeated exposure inhalation studies have been conducted in a wide range of laboratory animal species, and different exposure conditions. Many have focused on investigating mucociliary clearance, and the effects of varying humidity (DFG, 2001). The most useful is a 28-day study in rats (Kilgour, 2000). Groups of 15 female Wistar-derived rats were exposed nose-only for 6 hours/day, 5 days/week for up to 28 days to 0, 0.3, 1.38, and 5.52 mg/m^3 sulphuric acid mist (50% aqueous solution) (exposure concentrations refer to sulphuric acid content). The mass median diameters of the acid aerosols were 0.62, 0.83, and 0.94 µm for the low, mid and high exposure groups respectively. The key target was the upper respiratory tract. Histopathological examinations identified no lesions in the lungs or nasal passages, but significant exposure-related changes were found in the larynx. At 28 days, squamous epithelial metaplasia in the larynx was observed in 6/10 animals at 0.3 mg/m^3 (minimal grade) and in all animals at the two higher exposure levels; the severity was graded as slight to moderate at 1.38 mg/m^3 and moderate to marked at 5.52 mg/m^3. In addition, minimal to moderate laryngeal metaplasia was observed in 6/10 animals exposed to 5.52 mg/m^3. At the highest exposure level the lesion was still apparent (graded minimal to slight) in animals killed 4 and 8 weeks after exposure. These histological observations were paralleled by a statistically significant increase in tritiated thymidine uptake, indicating increased cell proliferation, in the laryngeal epithelium of animal exposed to 1.38 and 5.52 mg/m^3 H_2SO_4, but not at 0.3 mg/m^3.

Although the changes seen at the lowest exposure concentration (0.3 mg/m3) were slight, and could be regarded as an adaptive response, these observations suggest that there may be a risk of respiratory tract epithelial changes following longer-term repeated exposure at 0.3 mg/m^3 which are of concern when viewed in the context of observations of laryngeal cancer (see below).
Of the available studies in rabbits, in the longer term investigations animals were exposed to 0 or 0.25 mg/m^3 (MMAD 0.3 µm) for 1 hour/day, 5 days/week for 1 year (Gearhart and Schlesinger 1986, 1988, 1989). Relative to the controls, the exposed rabbits showed decreased mucociliary clearance, decreased airways diameter, increased secretory cell count, and increased bronchial reactivity. There were no histopathologically-observable signs of respiratory tract irritation. Similar results were obtained when rabbits were exposed to 0.125 mg/m^3 for 2 hours/day for 1 year (Schlesinger et al, 1992).

In a 20-month continuous exposure study in primates, groups of cynomolgus monkeys were exposed to 0, 0.38, 0.48, 2.43, and 4.79 mg/m^3 (MMAD 0.54-3.6 µm) (Alarie et al, 1973). This study provided evidence of enhanced toxicity with increased particle

size. When exposed to particles with MMAD 0.54 µm, a concentration of 0.48 mg/m^3 produced no histopathologically-observable lesions in the lungs. In contrast, with larger particles of MMAD 2.15 µm, the lowest concentration tested, 0.38 mg/m^3 produced mild hyperplasia in the bronchiolar epithelium, and slight wall thickening in the bronchioles and alveoli. At 4.79 mg/m^3 (MMAD 0.73 µm) there was marked hyperplasia of the bronchiolar epithelium and marked bronchiole wall thickening. In addition, an increased respiration rate was observed. At 2.43 mg/m^3 (MMAD 3.6 µm) there was moderate hyperplasia of the bronchiolar epithelium, marked wall thickening of the bronchioles and alveoli, and increased respiratory rate.

Overall, no clear NOAEL is discernable from the available repeated exposure studies in experimental animals, with evidence of various respiratory tract effects in rats, rabbits and monkeys on repeated exposure to concentrations in the range 0.125 – 0.38 mg/m^3.

2.6. Genotoxicity

There are no human genotoxicity data in relation to exposure to H_2SO_4. Experimentally, the genotoxicity of H_2SO_4 has been poorly investigated, partially due to the difficulties in accommodating the acidity of the concentrated substance in many test systems.. Some information is available from in vitro systems: addition of H_2SO_4 and the subsequent reduction in pH did not increase the mutation frequency in an Ames test or in studies using N.crassa or S.cerevisiae (Cipollaro et al, 1986, IARC 1992). Positive responses have been reported in in vitro mutagenicity assays (CHO and L5178Ycells) (Morita et al, 1989, Brusick 1986). These are considered to be a consequence of the pH changes produced by the addition of H_2SO_4. There are no in vivo data.

2.7. Carcinogenicity

The incidence of cancer has been investigated in H_2SO_4 exposed workers from a number of industries, including the production of isopropanol and ethanol, steel pickling, battery manufacture, soap manufacture and H_2SO_4 production (DFG, 2001, IARC 1992). About 20 epidemiology studies are available, of which four cohort and three case control studies are considered suitable for inclusion in an assessment of the carcinogenic potential in humans. Three cohort studies provided evidence of an approximate doubling of the risk of laryngeal cancer in the H_2SO_4-exposed population, and for only one was the risk less than 1.0. In addition, two out of three nested case-control studies gave increased a relative-risk for laryngeal cancer of around 7-13. There are some weaknesses in these studies, for example the analysis does not take account of potential confounders such as smoking, or the information on H_2SO_4 exposure is uncertain, but it is considered that there is adequate evidence for concluding that occupational exposure to H_2SO_4 is associated with an increased risk of cancer of the larynx. The incidences of laryngeal cancer of more than 2-5 are above the maximum risk of 1.75 estimated for smoking and alcohol consumption and can therefore not result from these confounders only. From the relatively poor quality of the exposure data presented in these studies and the recently recognised difficulties with the analytical techniques used to monitor sulphuric acid mist exposures in the past it is not possible to associate the increase in cancer incidence with particular exposure levels.

The IARC (1992) conclusion was that "There is sufficient evidence that occupational exposure to strong-inorganic-acid mists containing sulphuric acid is carcinogenic" (IARC 1992). In all those studies, sulphuric acid mists were the commonest exposure.

The presumed mechanism by which laryngeal cancer arose in these groups of workers is chronic inflammation of the epithelium in this region, caused by the acidity of sulphuric acid aerosols. This hypothesis links with the findings of the rat inhalation study of Kilgour et al (2000). A threshold would apply to this presumed carcinogenic mechanism, that being the dose at which the buffering capacity of the epithelial cells is overwhelmed and a significant fall in cellular pH occurs.

There have been some later cancer epidemiology studies. In some 2,500 men employed since 1950 in two battery plants and two steel works in Britain and followed up to 1993, mortality from all cancers and from cancer of the larynx and of the lung was less than in the national comparable population. When cases of upper aero digestive cancer (no. 15) within the cohort where compared with age matched controls (five per case) from the same plant, an increased risk, although statistically non significant, was apparent in those who had worked for at least five years in jobs where exposure to sulphuric or hydrochloric acid was likely to have exceeded 1mg/m^3 (Coggon et al. 1997). In another study, within the context of a large case-control study of multiple cancer sites and hundreds of occupational exposures, oesophageal cancer, particularly squamous cell carcinoma, appeared to be associated to a handful of occupational exposures, particularly sulphuric acid and carbon black (Parent et al. 2000). In a further study, a cohort of men employed for at least 1 year in an Italian sulphuric acid manufacturing plant was examined between 1962 and 1997. Overall mortality and cancer mortality were lower than in the regional comparable population. The relative risk for laryngeal cancer was 1.3 (95%CI 0.5-3.5) and for lung cancer it was 0.83. Most of the workers had previous exposure in mining (Pesatori et al. 2002).

A range of non-standard animal carcinogenicity studies are available. There was no increase in respiratory tract tumours amongst hamsters exposed by inhalation to 100 mg/m^3, 6 hours/day, 5 days/week for life, and no evidence for tumour-promoting activity when co-administered with benzo(a)pyrene (US EPA, 1978). It is noted that this exposure level is close to the 4-hour LC_{50} value obtained in other animal species suggesting that hamsters may be resistant to H_2SO_4 respiratory tract toxicity. In rats, repeated intratracheal administration produced a higher number of forestomach and oesophageal tumours, and tumours of the respiratory tract, lymphomas, and other unspecified tumours, compared to controls (Uleckiene and Griciute, 1997). However, the differences between test and control groups for any particular tumour type were not statistically significant and in view of this and other methodological weaknesses in the study no particular emphasis should be placed on the results. Similar comments apply to the work of the same authors in which rats received once-weekly oral gavage administration of 0.6% H_2SO_4 for the lifespan of 30 male and 30 female rats, where a higher (but not statistically significant) incidence of forestomach or oesophageal tumours (32% compared to 18% in controls) was noted (Uleckiene and Griciute, 1997). Two other studies provided little useful information on carcinogenic potential: one involved the study of respiratory tract tumours in mice following oral gavage administration; the respiratory tract is an unlikely target tissue for this route of exposure and indeed no tumours as a result of sulphuric acid administration was seen (Uleckiene and Griciute, 1997). The other employed a total of just 6 exposures (Swenburg and Beauchamp, 1997). No increases in tumour incidence were noted in this study. Overall, the experimental animal data provide little useful or reliable information on the carcinogenic potential of sulphuric acid.

2.8. Effects on reproduction

There are no human data in relation to reproductive effects of H_2SO_4 mists and there are no animal data on the potential effects on fertility. However, there are no grounds

to anticipate that such effects might occur because the protons and sulphate moieties would enter the general pool of these ions in vivo. In developmental studies in which mice and rabbits were exposed to 0, 5 or 20 mg/m^3 H_2SO_4 by inhalation during gestation, the only effect seen was the trivial finding of an increased incidence of non-ossified areas of the skull of rabbits at the highest exposure level (Murray et al, 1979). This was observed at an exposure level associated with significantly reduced maternal bodyweight. Mice were unaffected. Overall, H_2SO_4 is not considered to have the potential to cause specific reproductive toxicity in the developing fetus.

3. Recommendation

Concern about the toxicity of sulphuric acid in the workplace atmosphere is focussed on its potential, as an inhaled aerosol, to exert local effects on the respiratory tract, as a consequence of low pH. Such effects can be manifested as sensory irritation of nerve endings, acute or longer term inflammation at various sites along the length of the respiratory tract epithelium, and ultimately the possibility of tumour formation in the respiratory tract, believed to be a consequence of sustained tissue inflammation and repair processes. Human carcinogenicity data and the findings of a recent 28-day inhalation study in rats suggest that the larynx is a site of particular concern, in relation to epithelial inflammation, damage and ultimately cancer.

The identification of a clear NOAEL for this range of potential respiratory tract effects is difficult, from the available data. However, the recent 28-day inhalation study in rats (using a 50% sulphuric acid aerosol) provides evidence of slight changes in the laryngeal epithelium at the lowest concentration tested, 0.3 mg/m^3 Other experimental studies in a range of animal species suggest respiratory tract effects on repeated exposure to concentrations around 0.3 mg/m^3, with the possibility of effects of some health significance even at concentrations down to about 0.1 mg/m^3.

Taking into account the overall database, and with the concern for potential human carcinogenicity in mind, SCOEL concluded that long-term exposure should be maintained below 0.1 mg/m^3 in order to provide sufficient reassurance of avoidance of possible adverse consequences for the respiratory tract epithelium. Hence SCOEL recommends an 8h TWA limit of 0.05 mg/m^3 in order to satisfy this requirement. SCOEL appreciates that the reliable measurement of exposures at and around the limit value proposed is challenging. In some circumstances there might be interference from sulphate salts also present in the atmosphere. However, from the most recent evidence presented to SCOEL and from the assessment made in the Annex it appears that there are measurement techniques available that are compatible with the proposed limit.

In terms of health protection, SCOEL considered that it would be desirable to recommend a STEL of 0.1 mg/m^3 to avoid short-term irritant effects. However, at present there is no available measurement method which can accommodate a short-term limit at this value (see Annex)

There is no evidence that H_2SO_4 can penetrate undamaged skin to cause any signs of systemic toxicity, hence there is no requirement for a 'Sk' notation.

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Annex 1: Measurements of sulphuric acid

Analytical aspects to determine sulphuric acid at the values recommended by SCOEL

This issue was firstly partially considered in the SCOEL/INF/523 and in the SCOEL/INF/595 documents concerning analytical methods used in Germany to determine sulphuric acid. A summary of the documents above mentioned together with the information gathered from other methods currently used follows.
Table 1 summarises the Limit of Detection (LOD) for each method examined (i.e. NIOSH, OSHA ID-113, OSHA ID-165SG, BIA and BASF) expressed in the same units that the established OELs by the SCOEL for sulphuric acid (TWA = 0.05 mg/m^3 and STEL = 0.1 mg/m^3), in which the compliance with the OELs is made as well.

TABLE 1. COMPARISON BETWEEN LOD AND OELs FOR SULPHURIC ACID

METHOD	LOD AND SAMPLING TIME	EVALUATION	COMPLIANCE WITH THE SCOEL OELs
NIOSH 7903	0.02 mg/m^3;1.6h sampling time 0.12mg/m^3;15min. sampling time	VALIDATED	No * No *
OSHA ID-113	0.01mg/m^3; 4h sampling time 0.17mg/m^3; 15 min. sampling time	PARTIALLY	Yes No *
OSHA ID-165SG	0.003 mg/m^3; 8h sampling time 0.083 mg/m^3; 15min. sampling time	PARTIALLY	Yes No *
BIA (quartz fibre filter)	0.01 mg/m^3 ; 8h sampling time 0.5 mg/m^3 ; 15 min. sampling time	VALIDATED	Yes No *
BASF (absorbent liquid)	0.04 mg/m^3 ; 2h sampling time 0.3 mg/m^3 ; 15 min. sampling time	VALIDATED	No * No *

* To conform to European standard (EN 482, 1994) the LOD has to be less than 20% of the OEL, however a minimum of 10% is recommended.

As can be seen, the two OSHA methods and the BIA method, can be used to determine the time-weighted-average (TWA), however, the exposure peaks cannot be determined to assess the short time exposure level (STEL) because, in accordance with the occupational exposure recommendation of the SCOEL document SCOEL/SUM/105 final (November, 2002) the LOD must be less than 0.01 mg/m^3.

Limitations and interferences of the methods

The particulate salts of sulphates present will give a positive interference. It is not possible to distinguish the sulphate concentration from the sulphuric acid, because with the ionic chromatographic procedure there is no possibility to separate them, therefore a correction factor must be applied with the knowledge of the sulphate concentration. Otherwise, measurements in environments containing other sulphate compounds may produce interference thus, that sulphuric acid can give apparent higher sulphuric acid measurements then the real concentration.

When both sulphur trioxide and sulphuric acid are present, a monitoring system must be used, to detect the aerosols and the vapours. Some NIOSH, OSHA and BASF methods are not suitable for inhalable aerosols while other German, French and ISO methods are suitable and sufficiently sensitive.

When additional organic sulphur compounds are present, such as mercaptans, carbon disulfide or xanthogenates, no validated method with the necessary requirements is available.

Conclusion

On workplaces with exclusively sulphuric acid mists, no problems exist to measure the 8h-shift-concentrations, but it is not possible to monitor a 15 minute period with the necessary Limit of Detection (LOD).
When deciding upon an appropriate exposure monitoring method, the competent person must select a methodology which takes account of both limitations and interferences.

Annex 2: Sampling aspects, December 2012

The adverse effects of inhaled sulphuric acid particles have been examined in several animal species. A common finding is that adverse respiratory effects of sulphuric acid are dose- and particle size-dependent (ACGIH 2004).

Critical effects of occupational exposure to sulphuric acid are on bronchial mucociliary clearance and lung function, as well as sensory irritation of the eyes and the upper respiratory tract. These effects are seen at about 0.1 mg/m^3. At somewhat higher levels (about 0.2 mg/m^3), tooth erosion and pathological changes in nasal mucosa have been reported in exposed workers (NEG 2009).

Cellular changes of the respiratory tract epithelium have been observed in animals after repeated exposures in the range 0.125–0.38 mg/m^3 (Grasel 2003).

Epidemiologically, an excess risk of laryngeal cancer has been found among workers exposed to strong inorganic acid mists containing sulphuric acid. The mode of action of laryngeal cancer after acid mist exposure seems to be secondary to the local airway irritation by the acid (NEG 2009).

In total, it is plausible that damage to respiratory epithelium and cancer probably does not occur below exposure levels that affect mucociliary clearance and cause irritation.

Compared to experimental animals, humans have larger airways and a more symmetrical upper bronchial airway branching pattern. In addition, humans do considerable oral breathing, thus bypassing nasal airways. These differences contribute to a greater amount of upper bronchial airway particle deposition in humans, and there is a tendency to greater deposition near airway bifurcations (Bråtveit 2004).

Sulphuric acid forms metastable aerosols, the particle size of which being changing with humidity. Under usual conditions in ambient air, aerosol particle diameters are in the range between 0.3 and 0.6 µm, and in mists in the range from 10 to 15 µm (DFG 2001).

Exposures are poorly described in the available epidemiological studies. Measurements vary with the use of different sampling methods and are subject to interferences in consequence of the presence of inorganic and organic sulphur compounds.

Based upon lead acid battery plant studies, the particle size of sulphuric acid aerosols is generally less than 10 µm (Gamble *et al* 1984a, Gamble *et al* 1984b, Jones and Gamble 1984). However, sulphuric acid is highly soluble in water and readily absorbed from the upper respiratory tract following inhalation exposure. In the warm, humid conditions of the human upper respiratory tract, there is a likely increase in particle size of inhaled aerosols, because of the hygroscopic nature of sulphuric acid. Larger droplets (10-15 µm) deposit in the nose and smaller droplets (1-10 µm) would reach deeper into the respiratory tract (larynx, trachea and bronchi).

A measurement based on the thoracic value would protect against the central airway effects (i.e. clearance and pulmonary function changes), as well as laryngeal cancer associated with exposure to sulphuric acid aerosols (ACGIH 2004), while the use of an inhalable fraction would also protect from acute or longer term

inflammation along the respiratory tract epithelium, and ultimately the possibility of tumour formation in the respiratory tract, believed to be a consequence of sustained tissue inflammation and repair processes.

Based on these considerations, SCOEL recommends sampling the inhalable fraction.

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