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

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Employment,
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Table of Contents

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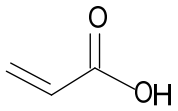
8-hour TWA:	10 ppm (29 mg/m^3)
STEL (1-min):	20 ppm (59 mg/m^3)
Notation:	None

Substance identification

Chemical name:	Acrylic acid
Synonyms:	2-propenoic acid, vinylformic acid
CAS No.:	79-10-7
EINECS No.:	201-177-9
Molecular formula:	$C_3H_4O_2$
Structural formula:	
Molecular weight:	72.06 g/mol
Conversion factor (25 °C):	1 ppm = 2.947 mg/m^3; 1 mg/m^3 = 0.339 ppm

EU Classification:

Flam. Liq. 3	H226	Flammable liquid and vapour
Acute Tox. 4 *	H332	Harmful if inhaled
Acute Tox. 4 *	H312	Harmful in contact with skin
Acute Tox. 4 *	H302	Harmful if swallowed
Skin Corr. 1A	H314	Causes severe skin burns and eye damage
Aquatic Acute 1	H400	Very toxic to aquatic life
STOT SE 3, C ≥ 1 %	H335	May cause respiratory irritation

This document is largely based on the EU-RAR (2002) and the references therein, along with some additional more recent published studies identified using the on-line database PubMed.

Physico-chemical properties

At 20 °C and 1 013 hPa, pure acrylic acid is a clear colourless liquid with an irritating acrid odour. The average odour threshold of acrylic acid in air is between 0.20–3.14 mg/m^3 (0.067 and 1.047 ppm). It is miscible with water and most organic solvents (IPCS 1997). Acrylic acid is flammable and combustible. The melting temperature is 14 °C and the boiling temperature is given as 141 °C. Acrylic acid has a flash point of 48–55 °C and the vapour pressure is 3.8 hPa at 20 °C. The pKa is given as 4.25 (EU RAR).

1. Occurrence/use and occupational exposure

The annual production volume of acrylic acid in the EU is estimated to be about 810 000 tonnes. About 20 000 tonnes are imported and at least 15 000 tonnes are exported outside the EU (EU RAR). The annual consumption of acrylic acid within the EU is about 830 000 tonnes. The market was reported to be undergoing rapid growth during the 1990s.

Acrylic acid is an industrial intermediate used to produce polyacrylate directly or polymerised via the intermediate stage of an acrylate ester. It is also used as an ingredient in products such as adhesives, which may contain up to 10 % acrylic acid, paints, binding agents and printing inks. Occupational exposure may arise during the production and processing of acrylic acid in the chemical industry and in the manufacture of products containing acrylic acid. Exposure may also occur during the use of adhesives and other products containing acrylic acid, during the decomposition of photoresistant materials with UV light (e.g. during the production of integrated circuits) and during the flame removal of paints.

1.1. Methods of exposure monitoring and analysis

1.1.1. Concentrations of acrylic acid in air

OSHA have published a partially validated method (OSHA Method PV2005, July 1996), which at a flowrate of 0.1 l/min during 4 hours onto Anasorb 708 tubes detects 0.2 µg.

The method proposed by Zanella *et al* (1999) also using diffusion denuder tubes with a subsequent HPLC technique reports detection of 2.9 µg/m^3 for a sample of 15 l (30 min at 0.5 l/min), which corresponds to an absolute detectable amount of 0.0435 µg.

Since a 1-min sampling time detects 5.9 µg (at 0.1 l/min - OSHA) or 29.4 µg (at 0.5 l/min - Zanella) a STEL of 20 ppm, which is restricted to 1 min, can be controlled. For practical reasons (very short sampling time), the use of a direct reading instrument (such as a photo ionisation detector) to assess the peak exposures to acrylic acid is recommended.

At the recommended OEL and STEL, no difficulties in measurement are expected.

1.1.2. Biological monitoring

There were no published methods of biological monitoring.

2. Health significance

2.1. Toxicokinetics

2.1.1. Absorption

Acrylic acid is almost completely absorbed via the inhalation and oral route. Dermal uptake was shown to be up to 26 %. The calculated nasal tissue dose for mice after inhalation of acrylic acid was 88 % higher than for rats (Barrow 1984). In rats, 97 % of the acrylic acid was deposited in the upper airways, indicating almost complete absorption after inhalation (Morris and Frederick 1995). Under similar exposure conditions, the uptake of acrylic acid by the human olfactory epithelium was predicted by a computational fluid dynamics and physiologically based pharmacokinetic dosimetry model to be 2- to 3-fold lower than that of rats (Frederick *et al* 1998, cited

in EU RAR 2002). Further refinements of the model predicted similar local deposition of acrylic acid in the olfactory epithelium of rats and humans at 4 and 25 ppm. The model used 43 parameters concerning respiration, tissue and mucus diffusivity, nasal surface and lumen, epithelia and mucus thickness, partition coefficients and gas phase mass transfer, amongst others, for rat and man (Andersen *et al* 2000, Frederick *et al* 2001). Some doubts were raised concerning the validity of the model, because important parameters (gas phase diffusivity, diffusivity in mucus, diffusivity in squamous epithelium and tissue diffusivity) were not measured but modelled (DFG 2006).

A simplified approach based on an even distribution of acrylic acid over the surface of the nasal epithelium and with the parameters listed in Table 1 led to the conclusion that the dose of the olfactory epithelium is about 1.6-fold higher in humans than in rats, also if the increased respiratory rate at the workplace is considered. However, as acrylic acid is highly water soluble it is mostly deposited anteriorly. In contrast to rats where a great part of the olfactory epithelium is located near the port of entry, the human olfactory epithelium is relatively small (Table 1) and located posteriorly, which implies that the dose of the olfactory epithelium in humans probably is not higher than that in rats (DFG 2006) and about 50 % lower than in mice (Barrow 1984).

Table 1. Dose and model parameters for the olfactory epithelium of rat and man at an exposure to 25 ppm acrylic acid (75 µg/l) (DFG 2006).

Parameter	Rat	Human
Minute volume	0.175 l/min, 250 g rat	20.8 l/min (10 m^3/8 h)
Acrylic acid inhaled/min	13.1 µg	1 563 µg
Surface of nasal epithelium	13.79 cm^2	245.9 cm^2
Surface of olfactory epithelium	6.72 cm^2	13.2 cm^2
Deposition in nose	97 %	ca. 50 %
Mean dose rate/cm^2 nasal epithelium (assuming constant nasal deposition)	0.92 µg/cm^2/min (13.1 µg/min/13.79 cm^2 × 0.97)	3.18 µg/cm^2/min (1 563 µg/min/245.9 cm^2 × 0.50)
Airstream over olfactory epithelium	ca. 15 %	ca. 7 %
Relative dose rate of acrylic acid for olfactory epithelium	1	1.6 (3.18/0.92 x 7 %/15 %)

The dermal absorption of acrylic acid is strongly dependent on the vehicle and pH of the solution. After dermal (occlusive) application of 5 mg acrylic acid/kg body weight to rats, cumulative absorption after 24 hours was dependent on the vehicle, with 22 % for acetone, 19 % for phosphate buffer pH 6 and 9 % for phosphate buffer pH 7.4. Under the condition of open application, 73 % of the acrylic acid (4 % solution) evaporated within 3 days, 21 % was absorbed and 6 % remained in the skin (EU RAR 2002).

Acrylic acid is rapidly and efficiently absorbed following ingestion (EU RAR).

Distribution

In a radiolabel study, Kutzman *et al* (1982) exposed rats (nose-only) to acrylic acid vapour for 1 minute. Ninety seconds after exposure, 18.3 % of the delivered dose remained in the rats. Approximately 28.0 % of this radioactivity was associated with the snout and 42.9 % of the radioactivity in the head. After 65 min, the activity in the snout was reduced to 8.1 %, and the radioactivity retained in liver and fat had increased markedly. Kutzman *et al* also administered an aqueous solution of radio-labelled acrylic acid by oral gavage to rats. The acrylic acid was rapidly absorbed and the radiolabel mainly expired as carbon dioxide within an hour of administration. The relative retention after 65 minutes was greatest in the liver. Approximately 6 % of the radiolabel was eliminated in the urine within 65 minutes. The authors used the short-lived ^{11}C as radiolabel, casting some doubt on the validity of the data.

Metabolism and excretion

Acrylic acid is rapidly metabolised to carbon dioxide which is formed via acrylyl-CoA by the non-vitamin-B12-dependent pathway of mammalian propionate catabolism (EU RAR). High doses of acrylic acid leading to tissue damage cause the formation of small amounts of mercapturic derivatives. About 80 % of an ingested dose of acrylic acid is exhaled as carbon dioxide within 24 hours. The kidneys and liver may be major sites of acrylic acid metabolism (Black *et al* 1993). A small proportion of absorbed acrylic acid is eliminated as urinary metabolites. The major urinary metabolite is 3-hydroxypropionic acid (EU RAR). Epoxidised metabolites of acrylic acid were not detected (EU RAR).

2.2. Acute toxicity

Pure acrylic acid is a very reactive chemical substance that exhibits severe corrosive properties when it comes into direct contact with biological material. The toxicity of acrylic acid is strongly dependent on its concentration, both in air and in the aqueous solution.

2.2.1. Human data

There were no reports of acute acrylic acid poisoning in humans (EU RAR, IPCS 1997).

2.2.2. Animal data

The EU RAR (2002) cites an LC_{50} of 3 600 mg/m^3 (4-hour exposure) in a poorly reported study in rats. Exposure to high concentrations of acrylic acid is reported to produce irritation of the nasal mucosa, the upper and lower airways and the eyes, corneal opacities and dermal toxicity (IPCS 1997). Silver *et al* (1981) reported a dose-dependent decrease in respiratory frequency and minute volume in rats exposed for 1 hour to 300, 900 and 1 500 mg/m^3 (100, 300, 500 ppm). The reduction was approximately 10–15 % at 100 ppm.

In rats, reported oral LD_{50} values range from 140 to 1 400 mg/kg body weight.

Acute dermal toxicity is dominated by severe local corrosion. The influence on uptake and toxicity due to the corrosivity of acrylic acid has to be considered. Dermal LD_{50} values of 300 and 640 mg/kg body weight in rabbits have been reported for undiluted acrylic acid (EU RAR). An LD_{50} of 1 350 mg/kg body weight was reported for male rats in a study with a 10 % aqueous solution of acrylic acid (pH of 2.5) in which it was believed that effects could be specifically attributed to acrylic acid *per se*, rather than the corrosive effects of acidity (EU RAR).

2.3. Irritation

2.3.1. Human data

The EU RAR cites three cases of accidental occupational exposure to acrylic acid that resulted in two admissions to hospital for skin corrosion and one admission for respiratory irritation.

The lateralisation threshold, indicative of a sensory irritation via trigeminal stimulation, was determined in 72 male and female persons. The median was 31 ppm (5-percentile 13 ppm) (van Thriel *et al* 2006).The results cannot be extrapolated to an 8-hour exposure, but are useful for setting a STEL. However, the irritation effect observed during a few minutes does not necessarily mean that longer exposure times result in increasing sensitivities to this reaction. This is supported by the literature review by Shusterman *et al* (2006) about time effects in human sensory irritation, which describes non-linearities in the time effects. They either showed a plateau or there was a reversal of the effects over time.

2.3.2. Animal data

Animal tests have demonstrated that acrylic acid is severely irritating to the respiratory tract. Majka *et al* (1974) reported severe irritation of the bronchial mucosa, exudate into the bronchial lumen, macrophages in the vesicle lumen and focal intraparenchymal irritation of the lungs in rabbits exposed to 2 970 mg/m^3.

Acrylic acid is severely corrosive to the skin, and exposure of rabbit skin to a 10 % solution of acrylic acid caused skin irritation after 5 minutes of exposure (unpublished report cited by EU RAR).

Acrylic acid causes severe damage to the eye in animals with irreversible corneal opacity and scarring of the eyelid. The serious damage to eyes caused by acrylic acid is not due to the acidic properties of this chemical, because in another study, neutralising the acid still led to irreversible corneal opacity (EU RAR).

Signs of local irritation (nasal discharge) were seen after repeated exposure to 300 and 1 500 ppm acrylic acid (Gage et al 1970; see Section 2.5.5).

2.4. Sensitisation

2.4.1. Human data

Workers exposed to acrylic acid can develop contact dermatitis but there is no strong evidence of skin sensitisation. There are two case reports of individuals displaying a positive response to acrylic acid in patch tests (Fowler 1990, Daecke *et al* 1993). Negative results were found in 6 other patch tested workers (Conde-Salazar *et al* 1988) and regular testing of more than 450 production workers during the 1990s failed to find evidence of skin sensitisation (EU RAR). It is possible that the reported cases of sensitisation to acrylic acid were actually due to sensitisation to an impurity of acrylic acid (EU RAR).

Respiratory sensitisation has not been reported.

2.4.2. Animal data

Pure acrylic acid has not shown skin sensitising properties in animal sensitisation tests (EU RAR). Positive results in older studies may have been due to 2,3-di(acryloxy)

propionic acid, a strongly sensitising impurity (DFG 2006) nowadays not contained anymore in commercial samples (EU RAR). There were no data for inhalation.

2.5. Repeated dose toxicity

2.5.1. Human data

No human data were available concerning chronic health effects of acrylic acid exposure, despite the widespread industrial use. A study of occupational exposure to chemicals during the production of acrylic acid by Tucek *et al* (2002) did not specifically measure exposure to acrylic acid or investigate the effects of acrylic acid exposure. Workers were exposed to a wide range of other chemicals but measured concentrations of chemicals in the working atmosphere were generally low and no health effects were found that could be attributed solely to acrylic acid.

2.5.2. Animal data

Inhalation

In a 90-day OECD-guideline compliant study, rats and mice were exposed for 6 hours each day to concentrations of 0, 15, 75 or 225 mg/m^3 (0, 5, 25 or 75 ppm) acrylic acid (Miller *et al* 1981). There was a reduction in the mean body weight gain of female mice in the 75- and 225-mg/m^3 (25 and 75 ppm) exposure groups. There were no significant differences in organ weights, clinical chemistry parameters, urine analysis parameters or gross pathology that could clearly be related to exposure. Slight focal degeneration of the nasal olfactory epithelium was observed in rats at 225 mg/m^3 (75 ppm), but no effects were seen at 15 or 75 mg/m^3 (5 or 25 ppm). In mice, there was a clear exposure-related increase in focal degeneration of the olfactory nasal epithelium with lesions being found in all animals in the 225-mg/m^3 (75 ppm) exposure group. The lesions were described as very slight at 15 mg/m^3 (5 ppm; 1/10 males and 4/10 females).

A brief summary is given below of the other studies described in the EU RAR (2002) that were not OECD-guideline compliant.

In a 2-week inhalation study, Miller *et al* (1981) exposed F-344 rats and B6C3F1 mice (five of each sex per group) to concentrations of 0, 75, 225 or 675 mg/m^3 (0, 25, 75 or 225 ppm) acrylic acid vapour for 6 hours each day, 5 days per week. Significant decreases in body weight gain were seen in animals exposed to 675 mg/m^3 (225 ppm) together with a reduction of adipose tissue in females exposed to this concentration. Lesions of the nasal mucosa and focal squamous metaplasia of nasal tissue were observed in rats at 675 mg/m^3 (225 ppm). In mice, lesions of the nasal mucosa were observed in 2 out of 5 males and 4 of the 5 females at 75 mg/m^3 (25 ppm) and in all mice exposed to 675 mg/m^3 (225 ppm).

Female B6C3F1 mice (15 per group) were exposed to acrylic acid vapour concentrations of 0, 5 or 25 ppm (6 or 22 hours/day) or to 25 ppm (4.4 hours/day) for 15 days. At the end of the exposure, 10 animals were sacrificed. The other 5 animals were sacrificed after a 6-week recovery period. Clinical parameters were recorded. Histopathologically, only the nasal cavity was examined. Exposure to 5 ppm for 6 hours/day did not result in effects. Concentrations of 5 ppm for 22 hours/day as well as 25 ppm for 4.4 hours/day resulted in concentration- and time-dependent changes in the olfactory epithelium with atrophy, basal cell hypertrophy, necrosis and degeneration of the Bowman gland. The findings after the 22-hour/day exposure to 5 ppm as well as after the 4.4- and 6-hour exposures to 25 ppm were fully reversible after 6 weeks. However, exposure to 25 ppm for 22 hours/day resulted in limited

regions of olfactory epithelium being replaced with respiratory-like epithelium (respiratory metaplasia) (Lomax *et al* 1994, Rohm and Haas Company 1994).

Gage (1970) found no effects in rats exposed to 240 mg/m^3 (80 ppm) acrylic acid vapour, 6 hours each day, 5 days per week for 4 weeks, whereas rats exposed at 900 mg/m^3 (300 ppm) showed signs of nasal irritation, lethargy and reduced body weight gain. Four exposures to 4 500 mg/m^3 (1 500 ppm) for 6 hours, resulted in nasal discharge, lethargy, retarded weight gain and kidney congestion.

Barrow (1986) found a reduction in respiratory function in rats and mice after exposure to 225 mg/m^3 (75 ppm) acrylic acid vapour for 6 hours per day for 5 days. The cell proliferation in the olfactory epithelium of these animals was increased 17-fold in mice as compared to only 4-fold in rats (Swenberg *et al* 1987).

Dermal exposure

One study cited by the EU RAR observed no irritant effects in mice following long term application of 1% acrylic acid in acetone. Another study reported that the incidence and severity of skin irritation was greater following exposure to 4 % than to 1 % in acetone, implying that some effects were observed in the lower dose group.

Oral administration

The EU RAR (2002) summarises data from several studies. The NOAEL in two 90-day studies were 40 mg/kg/day in male rats and 83 mg/kg/day female rats. An oral dose of 150 mg/kg/day has been reported to cause severe damage to the mucosa of the stomach and higher doses were associated with premature deaths and tubular degeneration/necrosis in the kidneys.

Summary on repeated dose administration

The toxic effects of acrylic acid are dominated by its local irritation. Prolonged inhalation of acrylic acid adversely affects the olfactory epithelium with a LOAEL of 15 mg/m^3 (5 ppm) in mice. For rats, a local NOAEL of 25 ppm was obtained.

2.6. Genotoxicity

Bacterial mutation studies have given negative results, whereas tests with mammalian cells yielded mixed results. The EU RAR (2002) concluded that the mutagenic potential of acrylic acid was limited to clastogenicity. Most *in vivo* assays gave negative results, and taking account of data available for structurally related acrylic compounds, the EU RAR (2002) considered it unlikely that acrylic acid is mutagenic *in vivo*.

2.7. Carcinogenicity

There were no human data, and no inhalation experiments have been undertaken in animals. Acrylic acid esters are rapidly metabolised in the nasal tissues to acrylic acid and the corresponding alcohol, therefore inhalation studies with acrylic esters can be used to assess the local carcinogenic potential of the acid. *n*-Butyl, ethyl and methyl acrylate were not carcinogenic in inhalation studies with rats and mice (DFG 2006). In rats exposed to doses equivalent to a mean dose of 9, 31 or 88 mg/kg/day acrylic acid in drinking water for 26 months (males) or 28 months (females), no treatment-related clinical, haematological or histopathological changes were detected in comparison with the controls other than a slightly reduced water consumption in high-dose males (Hellwig 1993). No skin tumours or skin irritation were observed in two lifetime studies in mice receiving repeated dermal applications of acrylic acid (EU RAR).

Overall, acrylic acid did not cause cancer in animals following oral or dermal administration and it can be expected from the results with esters that the acid is also not carcinogenic after inhalation.

2.8. Reproductive toxicity

Inhalation

Rats exposed to 0, 120, 360 and 1 080 mg/m^3 (0, 40, 120 and 360 ppm) during days 6–15 of gestation (6 hours/day) showed a concentration-related reduction in food and water intake leading to a reduction in body weight gain from the lowest concentration. Irritation of the respiratory tract and eyes was observed in the highest concentration group. No effects on reproductive performance including any evidence of developmental toxicity were observed. The NOAEL for developmental toxicity was 360 ppm with minimal maternal toxicity at 40 ppm (Klimisch and Hellwig 1991).

Rabbits exposed to concentrations of 0, 75, 225 and 675 mg/m^3 (0, 25, 75 and 225 ppm) during days 6–18 of gestation (6 hours/day whole body) showed no treatment related effects on gestational parameters. Concentration-related clinical signs (perinasal/perioral wetness, nasal congestion, reduced body weight gain and food consumption) were seen in the 225- and 675-mg/m^3 (75 and 225 ppm) groups. The NOAEL for developmental toxicity was 225 ppm (Bushy Run Research Center 1993, Neeper-Bradley *et al* 1997).

Offspring of rats exposed for 6 hours each day, during days 6–20 of gestation, to 150, 300, 600 or 900 mg/m^3 (50, 100, 200 or 300 ppm) acrylic acid showed signs of developmental toxicity (reduced foetal body weight) at 300 ppm acrylic acid, in the presence of overt signs of maternal toxicity (reduced body weight gain). The NOAEL for developmental toxicity was 200 ppm (Saillenfait *et al* 1999).

Oral

No effects on fertility were observed in oral reproductive toxicity studies.

DePass *et al* (1983) exposed male and female rats to 0, 83, 250 or 750 mg/kg/day for 13 weeks (before mating and throughout gestation and lactation). Each male was mated with 2 females. Dose-related reductions in food and water consumption and in body weight gain were observed. A non-significant reduction in the fertility of males and females, number of live pups and number of pups weaned was seen in the 750-mg/kg/day group.

In an OECD-guideline compliant 2-generation study (Hellwig *et al* 1997), a NOAEL of 460 mg/kg/day was derived for effects on fertility in rats exposed to 0, 500, 2 500 and 5 000 ppm in drinking water (53, 240 and 460 mg/kg body weight, resp.). Toxicity in the parent animals was expressed as a reduction in food and drinking water consumption accompanied by reduced body weights and reduced body weight gain. A parental NOAEL for general toxicity of 240 mg/kg/day was reported in the F_0 generation, but for the F_1 generation, the NOAEL was 53 mg/kg/day (EU RAR).

Conclusions

Acrylic acid is not considered to be a reproductive toxicant. The NOAEL for developmental toxicity was 200 ppm in rats and 225 ppm in rabbits. The LOAEL for rats was 300 ppm and was associated with maternal toxicity. For toxicity to fertility, the NOAEL was 460 mg/kg body weight in rats.

3. Recommendation

Acrylic acid is rapidly absorbed following inhalation, skin contact or ingestion, and is mainly metabolised by oxidative pathways to carbon dioxide, which is eliminated in exhaled air. Acrylic acid is severely irritating to the respiratory tract, severely corrosive to the skin and causes severe damage to the eyes. Despite the widespread industrial use of acrylic acid, there have been no studies of the effects of workplace exposure. There is no evidence that acrylic acid is likely to cause cancer. The mutagenic potential of acrylic acid in *in vitro* assays appears to be limited to clastogenicity and it is unlikely that acrylic acid is mutagenic *in vivo* (EU RAR).

The toxic effects of acrylic acid are dominated by local irritation. The NOAEL for effects on the olfactory epithelium was 75 mg/m^3 (25 ppm) in rats (Miller *et al* 1981), but not established for mice (Lomax *et al* 1994). The LOAEL in mice was 15 mg/m^3 (5 ppm). In female mice, the NOAEL for systemic toxicity was 15 mg/m^3 (5 ppm) and the LOAEL 75 mg/m^3 (25 ppm) (Miller *et al* 1981).

The NOAEL for developmental toxicity was 200 ppm in rats and 225 ppm in rabbits (Saillenfait *et al* 1999).

As has been argued in the EU RAR, from comparison of the 2- and 13-week studies with acrylic acid and studies with methyl acrylate and methyl methacrylate, the "nasal irritation threshold for acrylic acid will not substantially change when extrapolation is made from experimentally-tested subchronic exposure to chronic exposure". There are clear species differences in the deposition rate between rats and mice. Calculations suggest that humans have approximately the same deposition rate for acrylic acid as rats. Therefore, the rat, and not the mouse, is the most appropriate model for extrapolation to humans. The starting point for the calculation of the OEL is therefore the subchronic NOAEL of 25 ppm in rats. Acrylic acid does not have to be metabolised to cause irritation, so interindividual differences in sensory irritation thresholds should be small. Therefore, an 8-hour TWA of 10 ppm is considered appropriate to protect workers from histological changes and irritation.

The recommended OEL should not be exceeded significantly as irritation is to be expected in a significant number of workers, which is supported by the study of van Thriel *et al* (2006) who reported a lateralisation threshold (beginning of irritation in volunteers) of 30 ppm. Therefore, a STEL for acrylic acid of 20 ppm is proposed, which should be limited to 1 min.

No measurement difficulties are foreseen at the recommended OEL and the STEL of 1 min.

Due to the corrosive properties, routine dermal exposure to undiluted acrylic acid is unlikely. From the systemic oral NOAEL of 40 mg/kg body weight and the reported dermal uptake of 26 % from 1 % solutions (non-irritant) in rats, the corresponding amount of a 1 % solution can be calculated as 1 120 g for a 70-kg human. This amount is very large so that prolonged exposure to non-irritant solutions should not lead to systemic intoxications.

A "skin" notation is therefore not warranted.

There is no evidence that pure acrylic acid can cause respiratory or skin sensitisation, thus a "sensitiser" notation is not warranted.

4. References

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