Recommendation from the Scientific Committee

for Occupational Exposure Limits

for Ethyl acrylate

8 hour TWA:	5 ppm (21 mg/m^3)
STEL (15 mins):	10 ppm (42 mg/m^3)
Additional classification:	none

Substance:

Ethyl acrylate $H_2C=CH-COOCH_2CH_3$

Synonyms : Acrylic acid, ethyl ester; ethyl propenoate, 2-propenoic acid, ethyl ester; ethoxycarbonylethylene
EINECS N° : 205-438-8
EEC N°: 607-032-00-X
CAS N° : 140-88-5
MWt : 100.13
Conversion factor (20°C, 101kPa) : 4.17 mg/m^3 = 1 ppm
EU Classification: F; R11 Highly inflammable
Xn; R20/21/22 Harmful by inhalation, in contact with skin and if swallowed
Xi; R36/37/38- Irritating to eyes, respiratory system and skin
R43 May cause sensitization

Occurrence/use:

Ethyl acrylate is a colourless, flammable liquid with an acrid penetrating odour. It has a MPt of -71.2°C, a BPt of 99.8°C, a vapour pressure of 3.9 kPa at 20°C, a vapour density of 3.5 times that of air and has a lower explosive limit of 1.8% in air. The odour threshold is about 0.4 ppb (0.001 mg/m^3).

Ethyl acrylate is used in the manufacture of workers based paints, textiles and paper coatings. It is one of the principal monomers used worldwide in the production of styrene-

based polymers, which can be used for medical and dental items. The production rate in the EU is in excess of 10,000 tonnes per annum.

Health Significance:

The oral LD_{50} for ethyl acrylate in rats was reported to be approximately 1020 mg/kg bw. The maximum lethal oral dose for rabbits was 280-420 mg/kg bw. The LC_{50} for rats, following a 4-hour inhalation exposure, ranged from 1000 to 2000 ppm. The dermal LD_{50} for rabbits is reported to be 1790 mg/kg bw. The lowest lethal dermal dose for rats has been reported to be 1800 mg/kg bw (ACGIH, 2001).

Following inhalation exposure, ethyl acrylate is hydrolysed by carboxylesterases to acrylic acid in the nasal cavity (Frederick *et al*., 1994). Resorption is higher in the upper respiratory tract than in the lower respiratory tract (Stott and McKenna, 1984). After oral administration (gavage) ethyl acrylate is rapidly absorbed and distributed into all major tissues of rats. The major route of excretion after oral application is exhalation of CO_2 (about 70% of the administered dose) followed by urinary excretion of mercapturic acids, degradation products of GSH conjugates (Ghanayem *et al.*, 1987).

Ethyl acrylate is irritating to the skin and mucous membranes of the eyes and respiratory passages (DFG, 1994; Potokar et al., 1985).

Single oral dosing of ethyl acrylate by gavage (100-400 mg/kg bw) produced oedema in rat forestomach and glandular stomach by direct acting irritation (Ghanayem *et al*., 1985). Repetitive dosing by gavage caused mucosal oedema associated with vesicle formation, mucosal hyperplasia, erosions or ulcers and inflammation (Ghanayem *et al*, 1986). Similar lesions were found in the nasal cavity of rats after inhalatory exposure (Miller *et al.*, 1985). During a long-term inhalation study in rats and mice (6h/d, 5d/w, for 27 months) reduced body weight gain was observed at exposure levels of 72 ppm (300 mg/m^3) and above. Histopathological changes in olfactory portions of the nasal mucosa were present at levels of 25 ppm (100 mg/m^3) and above. These microscopic exposure-related changes were concentration-dependent, primarily in terms of distribution of the lesions within the nasal cavity (see tables 1, 2). In a follow-up study with 5 ppm (21 mg/m^3; 6 h/d, 5 d/w, for 24 months) no treatment-related changes in the nasal mucosa were observed in rats or mice (NOAEL) (Miller *et al*., 1985).

Table 1 Histopathological changes (percentages of animals with indicated observations) in olfactory epithelium of Fischer 344 rats exposed to ethyl acrylate vapours (Miller *et al*., 1985)

Observations	Exposure Group (ppm)									
	Males					Females				
	0-A	0-B	25	75	225	0-A	0-B	25	75	225
Basal cell hyperplasia										
Slight	2	0	68	1	52	0	0	55	4	66
Moderate	0	0	9	99	18	0	0	16	96	9
Increased intraepithelia glands										
Slight	0	0	42	1	1	0	0	12	0	4
Moderate	0	0	7	97	46	0	2	17	100	71
Respiratory metaplasia										
Slight	0	2	13	12	10	0	3	4	56	7
Moderate	2	2	3	83	15	0	0	2	24	1
Diffuse atrophy	2	2	5	0	92	0	1	0	0	80
Multifocal mineralization	0	0	1	87	42	0	0	8	87	17

Table 2 Histopathological changes (percentages of animals with indicated observations) in olfactory epithelium of $B6C3F_1$ mice exposed to ethyl acrylate vapours (Miller *et al*., 1985)

Observations	Exposure Group (ppm)									
	Males					Females				
	0-A	0-B	25	75	225	0-A	0-B	25	75	225
Hyperplasia of submucosal glands										
very slight	42	26	4	1	1	28	39	3	0	0
slight	0	2	48	1	4	0	2	81	0	0
moderate	0	0	41	34	10	0	0	3	83	3
severe	0	0	0	61	83	0	0	0	14	95
Respiratory metaplasia of olfactory epithelium										
very slight	47	30	0	1	0	28	39	3	0	0
slight	0	3	56	1	1	0	2	81	0	0
moderate	0	2	41	36	10	0	0	3	83	3
severe	0	0	0	61	87	0	0	0	14	95

When ethyl acrylate was administered to F344 rats and $B6C3F_1$ mice chronically by gavage in doses of 100 or 200 mg/kg bw, squamous cell papillomas and carcinomas of the forestomach were observed (NTP, 1986). Results of further studies in rats indicate that the

forestomach neoplasia is correlated to extensive and sustained forestomach mucosal hyperplasia and cell proliferation (Ghanayem *et al.*, 1991, 1993, 1994) which may be caused due to severe depletion of critical cellular thiols, mainly glutathione (Gillette and Frederick, 1993; Frederick *et al.*, 1990). There was no evidence of carcinogenicity in either rats or mice after inhalatory exposure (Miller *et al*., 1985) or in mice after dermal exposure (DePass et al., 1984).

Ethyl acrylate did not induce mutations in bacteria *in vitro* (IARC, 1999). In mammalian cells ethyl acrylate was tested nearly always in the absence of exogenous metabolic activation. Small colony mutations were induced in L5178Y mouse lymphoma cells at the *tk* locus (Amtower *et al*., 1986; Dearfield *et al.*, 1991; McGregor *et al.*, 1988; Moore *et al.*, 1988, 1989) indicating clastogenic activity (Amtower *et al*., 1986) or cytotoxicity mediated by depletion of nonprotein sulfhydryls and mitochondrial membrane impairment (Ciaccio *et al.*, 1998). No mutations were found in Chinese hamster ovary cells at the *hprt* locus (Moore *et al.*, 1989, 1991). Ethyl acrylate was found to induce chromosomal aberrations in L5178Y mouse lymphoma cells (Moore *et al.*, 1988, 1989) and Chinese hamster ovary (Moore *et al.*, 1989) and lung cells (Ishidate *et al.*, 1981) *in vitro. In vivo*, micronuclei formation was observed in bone marrow of mice following intraperitoneal injection of ethyl acrylate (2 x 225 mg/kg bw) at doses which caused toxicity (Przybojewska *et al*., 1984) but this result could not be reproduced in another study with higher intraperitoneal doses (2 x 738 mg/kg bw or 2 x 812 mg/kg bw) (Ashby *et al.*, 1989). In splenocytes from mice given a single intraperitoneal dose of ethyl acrylate (1000 mg/kg bw) no chromosomal aberrations or sister chromatid exchanges were reported, but a weak increase in micronuclei formation was found (Kligerman *et al.*, 1991). No DNA strand breaks were found in the forestomach of rats given 4% ethyl acrylate by gavage (Morimoto *et al.*, 1990). After application of 12 µg ethyl acrylate on mouse skin three times a week for 20 weeks, no DNA strand breaks or micronuclei formation was detected in peripheral blood cells (Tice *et al.*, 1997).

Pregnant Sprague-Dawley rats were exposed to 0, 50, or 150 ppm ethyl acrylate for 6 h/day during days 6 through 15 of gestation. In the presence of maternal toxicity at 150 ppm (decreased body weight gain and food consumption, increased water consumption), a slight but not statistically significant increase in malformed fetuses was observed. At 50 ppm, there was neither maternal toxicity nor an adverse effect on the developing embryo or fetus (Murray et al., 1981). In a further developmental toxicity study Sprague-Dawley rats were exposed during days 6 to 20 of gestation to 25, 50, 100 or 200 ppm ethyl acrylate for 6 h/day. No treatment-related increases in embryo/foetal mortality or foetal malformations were observed. Foetal toxicity, indicated by reduced foetal body weight, was observed after exposure to 200 ppm ethyl acrylate in the presence of overt signs of maternal toxicity (Saillenfait *et al.,* 1999).

There are no human data available which are adequate for proposing occupational exposure limits. A concentration of 50 ppm has been cited as being irritating to the eyes, nose and throat of humans (Deichmann and Gerarde, 1969), but without reference to the original study.

Skin sensitisation and cross reactions have been reported (Fregert, 1978; Jordan, 1975; Opdyke, 1975; see also DFG, 2001: Casse *et al*., 1998; Condé-Salazar *et al.*, 1988; Estlander *et al.*, 1996; IVDK, 1999; Jagtman, 1998; Jordan, 1975; Kanerva *et al.*, 1988,

1989, 1992, 1993, 1996a, 1996b, 1997, 1998; Kiec-Swierczynska, 1996; Koppula *et al.*, 1995; Miranda-Romero *et al.*, 1998; Rustemeyer and Frosch, 1996; Schnuch *et al.*, 1998; Tucker and Beck, 1999). In some cases sensitization was induced by patch tests (Kanerva *et al.*, 1988). These findings are supported by positive results in an FCA test and in a Buehler test with ethyl acrylate in guinea pigs both with and without the use of adjuvant (Parsons and Baldwin, 1981; van der Walle *et al.*, 1982). A non-occlusive patch test and a maximization test, however, yielded negative results (Klecak, 1985; van der Walle *et al.*, 1982). Negative results were also reported in a murine Local Lymph Node Asssay and a Mouse Ear Swelling Test (Hayes and Meade, 1999). There are no data available for sensitizing effects on the respiratory passages.

In a prospective cohort study a group of 60 workers exposed to chemical substances in the production of acrylic acid, acrylic acid esters and acrylate dispersions, and 60 controls were followed up from 1992 to 1999. The average exposure period was 13±5 years. Exposure to acrylonitrile, n-butanol, butyl acrylate, ethyl acrylate, methyl acrylate, methyl methacrylate, toluene, and styrene was determined by personal passive dosimetry. The measured concentrations were generally low, occasionally exceeding maximum allowable concentrations. 80% of the samples from personal passive dosimetry showed ethyl acrylate concentrations below 0.2 mg/m^3 (0.05 ppm) and about 10% of the samples showed ethyl acrylate concentrations of 0.21 to 1.0 mg/m^3 (0.05 to 0.24 ppm). Maximal concentrations ranged over 10 mg/m^3 (2.4 ppm). The results of the clinical, haematological and biochemical examination of the workers have not revealed any marked differences between the exposed and control groups that could be attributable solely to the acrylate exposure (Tuček *et al.,* 2002). Due to low concentrations of ethyl acrylate, the study is not suitable for evaluating a concentration of more than 2.4 ppm ethyl acrylate.

Mortality from colon and rectum cancer has been reviewed in three cohorts working in 1933 to 1982 in two plants manufacturing and polymerizing acrylate monomers. The two cohorts with later dates of employment showed no excess mortality. In the earliest cohort, excess colon cancer seemed restricted to men employed in the early 1940s in jobs entailing the highest exposures to vapor-phase ethyl acrylate and methyl methacrylate monomer and volatile by-products of the ethyl and methyl methacrylate polymerization process. The excess mortality only appeared some two decades after the equivalent of three years' employment in jobs with the most intense exposures. A smaller elevation in colon cancer mortality also appeared in a low-exposure group in the early cohort. Rectal cancer mortality was elevated in the same categories that showed excess rates of colon cancer death. Because of the lower rates, the rectal cancer results are more imprecise (Walker *et al.*, 1991).

Recommendation:

The study of Tucek et al. (2002) shows that repeated exposure up to 2.4 ppm does not induce adverse effects in workers. Miller et al. (1985) established a NOAEL of 5 ppm (21 mg/m^3) and a LOAEL of 25 ppm^3 for slight to moderate hyperplasia and metaplasia of the nasal mucosa in rats and mice after 24 or 27 months of exposure with a steep increase of effects at 75 ppm. Given a higher sensitivity of rats and mice to irritating effects in the nasal cavity (DeSesso 1992) an uncertainty factor is not considered to be necessary for proposing a occupational exposure limit. An 8-hour TWA of 5 ppm (21 mg/m^3) is

recommended and a STEL (15 min) of 10 ppm (42 mg/m^3) is recommended based on a pragmatic approach of multiplying the TWA OEL by a factor of 2.

No "skin" notation was considered necessary.

Ethyl acrylate should be recognised as a skin sensitiser.

At the levels recommended, no measurement difficulties are foreseen.

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