

***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	<chem>H2C=CH-COOCH2CH3</chem>
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/m³).

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 LD₅₀ 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 LC₅₀ for rats, following a 4-hour inhalation exposure, ranged from 1000 to 2000 ppm. The dermal LD₅₀ 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 CO₂ (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/m³) and above. Histopathological changes in olfactory portions of the nasal mucosa were present at levels of 25 ppm (100 mg/m³) 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/m³; 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 B6C3F₁ 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 B6C3F₁ 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 Assay 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/m³) 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.

Key Bibliography:

ACGIH (American Conference of Governmental Industrial Hygienists) (2001). Ethyl acrylate.

Amtower, A.L., Brock, K.H., Doerr, C.L., Dearfield, K.L. Moore, M.M. (1986). Genotoxicity of three acrylate compounds in L5178Y mouse lymphoma cells. *Environ. Mutagen. Suppl.* 6, 4.

Ashby, J., Richardson, C.R., Tinwell, H. (1989). Inactivity of ethyl acrylate in the mouse bone marrow micronucleus assay. *Mutagenesis* 4, 283–285.

Casse, V., Salmon-Ehr, V., Mohn, C., Kalis, B. (1998). Dépigmentation durable secondaire à des tests positifs aux dérivés des méthacrylates. *Ann. Dermatol. Venereol.* 125, 56–57.

Ciaccio, P.J., Gicquel, E., O'Neill, P.J., Scribner, H.E., Vandenberghe, Y.L., (1998). Investigation of the positive response of ethyl acrylate in the mouse lymphoma genotoxicity assay. *Toxicol. Sci.* 46: 324–332.

Condé-Salazar, L., Guimaraens, D., Romero, L.V. (1988). Occupational allergic contact dermatitis from anaerobic acrylic sealants. *Contact Dermatitis* 18, 129–132.

Dearfield, K.L., Harrington-Brock, K., Doerr, C.L., Rabinowitz, J.R., Moore, M.M. (1991). Genotoxicity in mouse lymphoma cells of chemicals capable of Michael addition. *Mutagenesis* 6, 519–525.

DeSesso J (1992) The relevance to humans of animal models for inhalation studies of cancer in the nose and upper airways. *Quality Assurance: Good Practice, Regulation and Law* Vol 2, No 3,: 213-231

Deichmann, W.B., Gerarde, H.W. (1969). *Toxicology of Drugs and Chemicals*, p75, Academic Press, New York.

DePass, L.R., Fowler, E.H., Meckley, D.R., Weil, C.S. (1984). Dermal oncogenicity bioassays of acrylic acid, ethyl acrylate, and butyl acrylate. *J. Toxicol. Environ. Health* 14, 115–120.

DFG (Deutsche Forschungsgemeinschaft) (1994). *Occupational Toxicants, Critical Data Evaluation for MAK Values and Classification of Carcinogens*, Ethyl acrylate, Vol. 6, 217–229.

- DFG (Deutsche Forschungsgemeinschaft) (2001). Occupational Toxicants, Critical Data Evaluation for MAK Values and Classification of Carcinogens, Ethyl acrylate, Vol. 16, 41–46.
- Estlander, T., Kanerva, L., Kari, O., Jolanki, R., Mölsä, K. (1996). Occupational conjunctivitis associated with type IV allergy to methacrylates. *Allergy* 51, 56–59.
- Frederick, C.B., Hazelton, G.A., Frantz, J.D., (1990). The histopathological and biochemical response of the stomach of male F344/N rats following two weeks of oral dosing with ethyl acrylate. *Toxicol. Pathol.* 18, 247–256.
- Frederick, C.B., Udinsky, J.R., Finch, L. (1994). The regional hydrolysis of ethyl acrylate to acrylic acid in the rat nasal cavity. *Toxicol. Lett.* 70, 49–56.
- Fregert, S. (1978). Allergic contact dermatitis from ethylacrylate in a window sealant. *Contact Dermatitis* 4, 56.
- Ghanayem, B.I., Maronpot, R.R. Matthews, H.B. (1985). Ethyl acrylate-induced gastric toxicity. I. Effect of single and repetitive dosing. *Toxicol. Appl. Pharmacol.* 80, 323–335.
- Ghanayem, B.I., Maronpot, R.R. Matthews, H.B. (1986). Ethyl acrylate-induced gastric toxicity. III. Development and recovery of lesions. *Toxicol. Appl. Pharmacol.* 30, 576–583.
- Ghanayem, B.I., Burka, L.T., Matthews, H.B. (1987). Ethyl acrylate distribution, macromolecular binding, excretion, and metabolism in male Fisher 344 rats. *Fundam. Appl. Toxicol.* 9, 389–397.
- Ghanayem, B.I., Matthews, H.B., Maronpot, R.R. (1991). Sustainability of forestomach hyperplasia in rats treated with ethyl acrylate for 13 weeks and regression after cessation of dosing. *Toxicol. Pathol.* 19, 273–279.
- Ghanayem, B.I., Sanchez, I.M., Maronpot, R.R., Elwell, M.R., Matthews, H.B. (1993). Relationship between the time of sustained ethyl acrylate forestomach hyperplasia and carcinogenicity. *Environ. Health Perspect.* 101, Suppl 5, 277–279.
- Ghanayem, B.I., Sanchez, I.M., Matthews, H.B., Elwell, M.R. (1994). Demonstration of a temporal relationship between ethyl acrylate-induced forestomach cell proliferation and carcinogenicity. *Toxicol. Pathol.* 22, 497–509.
- Gillette, D.M., Frederick, C.B. (1993). Quantitation of an epithelial S-phase response in the rat forestomach and glandular stomach following gavage dosing with ethyl acrylate. *Toxicol. Appl. Pharmacol.* 122, 244–257.
- Hayes, B.B., Meade, B.J. (1999). Contact sensitivity to selected acrylate compounds in B6C3F₁ mice: relative potency, cross reactivity, and comparison of test methods. *Drug Chem. Toxicol.* 22, 491–506.
- IARC (1999). Ethyl acrylate. IARC Monogr. Eval. Carcinog. Risks. Hum., Vol. 71, Pt 3, 1447–1457.
- Ishidate, M., Sofuni, T. Yoshikawa, K. (1981). Chromosomal aberration test in vitro as a primary screening tool for environmental mutagens and/or carcinogens. *Gann Monogr. Canc. Res.* 27, 95–108.

- IVDK (Informationsverbund Dermatologischer Kliniken) (1999). Auswertung der zwischen 1989 und 1998 vom IVDK erfaßten Daten zu Ethylacrylat, 05.05.1999.
- Jagtman, B.A. (1998). Contact dermatitis from acrylates in an electrosurgical earthing plate. *Contact Dermatitis* 38, 280–281.
- Jordan, W.P. jr. (1975). Cross-sensitisation patterns in acrylate allergics. *Contact Dermatitis* 1, 13–15.
- Kanerva, L., Estlander, T., Jolanki, R. (1988). Sensitization to patch test acrylates. *Contact Dermatitis* 18, 10–15.
- Kanerva, L., Estlander, T., Jolanki, R. (1989). Allergic contact dermatitis from dental composite resins due to aromatic epoxy acrylates and aliphatic acrylates. *Contact Dermatitis* 20, 201–211.
- Kanerva, L., Estlander, T., Jolanki, R., Pekkarinen, E. (1992). Occupational pharyngitis associated with allergic patch test reactions from acrylics. *Allergy* 47, 571–573.
- Kanerva, L., Jolanki, R., Estlander, T. (1993). Accidental occupational sensitization caused by methyl acrylate. *Eur. J. Dermatol.* 3, 195–198.
- Kanerva, L., Estlander, T., Jolanki, R. (1996a). False negative patch test reaction caused by testing with dental composite acrylic resin. *Int. J. Dermatol.* 35, 189–192.
- Kanerva, L., Lauerma, A., Estlander, T., Alanko, K., Henriks-Eckerman, M.-L., Jolanki, R. (1996b). Occupational allergic contact dermatitis caused by photobonded sculptured nails and a review of (meth)acrylates in nail cosmetics. *Am. J. Contact Dermatitis* 7, 109–115.
- Kanerva, L., Estlander, T., Jolanki, R. (1997). 10 years of patch testing with the (meth)acrylate series. *Contact Dermatitis* 37, 255–258.
- Kanerva, L., Mikola, H., Henriks-Eckerman, M.-L., Jolanki, R., Estlander, T. (1998). Fingertip paresthesia and occupational allergic contact dermatitis caused by acrylics in a dental nurse. *Contact Dermatitis* 38, 114–116.
- Kiec-Swierczynska, M. (1996). Occupational allergic contact dermatitis due to acrylates in Lodz. *Contact Dermatitis* 34, 419–422.
- Klecak, G. (1985). The Freund's complete adjuvant test and the open epicutaneous test. *Curr. Probl. Dermatol.* 14, 152–171.
- Kligerman, A.D., Atwater A.L., Bryant, M.F., Erexson, G.L., Kwanyuen, P., Dearfield, K.L. (1991). Cytogenetic studies of ethyl acrylate using C57BL/6 mice. *Mutagenesis* 6, 137–141.
- Koppula, S.V., Fellman, J.H., Storrs, F.J. (1995). Screening allergens for acrylate dermatitis associated with artificial nails. *Am. J. Contact Dermatitis* 6, 78–85.
- McGregor, D.B., Brown, A., Cattanach, P., Edwards, I., McBride, D., Riach, C., Caspary, W.J. (1988). Responses of the L5178Y tk⁺/tk⁻ mouse lymphoma cell forward mutation assay: III. 72 coded chemicals. *Environ. Mol. Mutagen.* 12, 85–154.
- Miller, R.R., Young, J.T., Kociba, R.J., Keyes, D.G., Bodner, K.M., Calhoun, L.L. Ayres, J.A. (1985). Chronic toxicity and oncogenicity bioassay of inhaled ethyl acrylate in Fischer 344 rats and B6C3F1 mice. *Drug Chem. Toxicol.* 8, 1–42.

- Miranda-Romero, A., Martínez, M., Sanchez-Sambucety, P., Aragoneses, H., Munoz, C.M. Garcia (1998). Allergic contact dermatitis from the acrylic adhesive of a surgical earthing plate. *Contact Dermatitis* 38, 279–280.
- Moore, M.M., Amtower, A., Doerr, C.L., Brock, K.H., Dearfield, K.L. (1988). Genotoxicity of acrylic acid, methyl acrylate, ethyl acrylate, methyl methacrylate, and ethyl methacrylate in L5178Y mouse lymphoma cells. *Environ. Mol. Mutagen.* 11, 49–63.
- Moore, M.M., Harrington-Brock, K., Doerr, C.L., Dearfield, K.L. (1989). Differential mutant quantitation at the mouse lymphoma tk and CHO hgpert loci. *Mutagenesis* 4, 394–403.
- Moore, M.M., Parker, L., Huston, J., Harrington-Brock, K., Dearfield, K.L. (1991). Comparison of mutagenicity results for nine compounds evaluated at the hgpert locus in the standard and suspension CHO assays. *Mutagenesis* 6, 77–85.
- Morimoto, K., Tsuji, K., Osawa, R., Takahashi, A. (1990). DNA damage test in forestomach squamous epithelium of F344 rat following oral administration of ethyl acrylate. *Eisei Shikenjo Hokoku* 108: 125–128.
- Murray, J.S., Miller, R.R., Deacon, M.M., Handley, T.R., Hayes, W.C., Rao, K.S., John, J.A. (1981). Teratological evaluation of inhaled ethyl acrylate in rats. *Toxicol. Appl. Pharmacol.* 60, 106–111.
- NTP (National Toxicology Program) (1986). Carcinogenesis studies of ethyl acrylate in F344/N rats and B6C3F₁ mice (gavage studies). NTP TR 259, NIH Publication No. 87-2515, U.S. Department of Health and Human Services, Public Health Services, National Institute of Health, Research Triangle Park, NC, USA
- Opdyke, D.L.J. (1975). Ethyl acrylate. *Fd. Cosm. Toxicol. suppl.* 13, 801-802.
- Parsons, R.D., Baldwin, R.C. (1981). Delayed contact hypersensitivity of guinea pigs to ethyl acrylate. *Toxicologist* 1, 17.
- Potokar, M., Grundler, O.J., Heusener, A., Jung, R., Mürmann, P., Schöbel, C., Suberg, H. Zechel, H.K. (1985). Studies on the design of animal tests for the corrosiveness of industrial chemicals. *Fd.Chem. Toxicol.* 23, 615–617.
- Przybojewska, B., Dziubaltowska, E and Kowalski, Z. (1984). Genotoxic effects of ethyl acrylate and methyl acrylate in the mouse evaluated by the micronucleus test. *Mutat. Res.* 135, 189–191.
- Rustemeyer, T., Frosch, P.J. (1996). Occupational skin diseases in dental laboratory technicians. (I). Clinical picture and causative factors. *Contact Dermatitis* 34, 125–133.
- Saillenfait, A.M., Bonnet, P., Gallissot, F., Protois, J.C., Peltier, A., Fabries, J.F. (1999). Relative developmental toxicities of acrylates in rats following inhalation exposure. *Toxicol Sci* 48, 240–254.
- Schnuch, A., Uter, W., Geier, J., Frosch, P.J., Rustemeyer, T. (1998). Contact allergies in healthcare workers. Results from the IVDK. *Acta Derm. Venereol. (Stockh)* 78, 358–363.

- Stott, W.T., McKenna, M.J. (1984). The comparative absorption and excretion of chemical vapors by the upper, lower, and intact respiratory tract of rats. *Fundam. Appl. Toxicol.* 4(4), 594–602.
- Tice, R.R., Nylander-French, L.A., French, J.E (1997). Absence of systemic in vivo genotoxicity after dermal exposure to ethyl acrylate and tripropylene glycol diacrylate in Tg.AC (v-Ha-ras) mice. *Environ. Mol. Mutagen.* 29, 240–249.
- Tuček, M., Tenglerova, J., Kollarova, B., Kvasnickova, M., Maxa, K., Mohyluk, I., Svandova, E., Topolcan, O., Vlasak, Z., Cikrt, M. (2002). Effect of acrylate chemistry on human health. *Int. Arch. Occup. Environ. Health* 75, Suppl 1, 67–72.
- Tucker, S.C., Beck, M.H. (1999). A 15-year study of patch testing to (meth)acrylates. *Contact Dermatitis* 40, 278–279.
- van der Walle, H.B., Klecak, G., Geleick, H., Bensink, T. (1982). Sensitizing potential of 14 mono (meth) acrylates in the guinea pig. *Contact Dermatitis* 8, 223–235.
- Walker, A.M., Cohen, A.J., Loughlin, J.E., Rothman, K.J., DeFonso, L.R. (1991). Mortality from cancer of the colon or rectum among workers exposed to ethyl acrylate and methyl methacrylate. *Scand. J. Work Environ. Health* 17, 7–19.