2-ETHYLHEXYL ACRYLATE

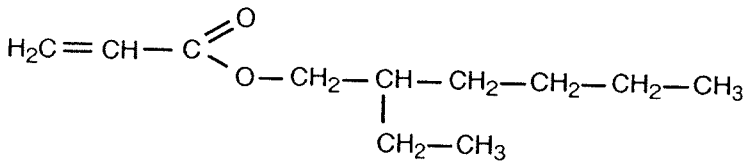
1. Exposure Data

1.1 Chemical and physical data

1.1.1 *Nomenclature*

Chem. Abstr. Serv. Reg. No.: 103-11-7
Deleted CAS Reg. No.: 78733-32-1; 84948-57-2; 93460-77-6
Chem. Abstr. Name: 2-Propenoic acid, 2-ethylhexyl ester
IUPAC Systematic Name: Acrylic acid, 2-ethylhexyl ester
Synonyms: 2-Ethylhexyl 2-propenoate

1.1.2 *Structural and molecular formulae and relative molecular mass*



$C_{11}H_{20}O_2$ Relative molecular mass: 184.28

1.1.3 *Chemical and physical properties of the pure substance*

(*a*) *Description*: Colourless liquid (Hoechst Celanese Corp., 1992, undated)
(*b*) *Boiling-point*: 213.5 °C (Hoechst Celanese Corp., 1992, undated)
(*c*) *Melting-point*: –90 °C (Ohara *et al.*, 1985)
(*d*) *Density*: Specific gravity, 0.8865 at 20 °C/20 °C (Hoechst Celanese Corp., 1992, undated)
(*e*) *Spectroscopy data*: Infrared, nuclear magnetic resonance and mass spectral data have been reported (Weast & Astle, 1985; Sadtler Research Laboratories, 1991).
(*f*) *Solubility*: Slightly soluble in water (0.01 wt% at 20 °C); soluble in alcohols, ethers and many organic solvents (acetone, benzene, ethyl ether, heptane, methanol, carbon tetrachloride) (Union Carbide Corp., 1982)
(*g*) *Volatility*: Vapour pressure, 0.14 mm Hg [19 Pa] at 20 °C; relative vapour density (air = 1), 6.4 at 20 °C (Hoechst Celanese Corp., 1992, undated)
(*h*) *Stability*: Flash-point, 92 °C (open cup); rapid, uncontrolled polymerization can cause explosion (Tyler, 1993)

(*i*) *Octanol–water partition coefficient (P)*: log P, 3.67 (Beratergremium für umweltrelevante Altstoffe, 1993); 4.32 (Tyler & Smock, 1993)

(*j*) *Conversion factor*: $mg/m^3 = 7.54 \times ppm$[a]

1.1.4 *Technical products and impurities*

2-Ethylhexyl acrylate is available as a commercial product with the following specifications: assay, 99.5 wt% min. (this value includes up to 0.4% 2-ethyl-4-methylpentyl acrylate, which is equivalent to 2-ethylhexyl acrylate in reactivity and performance in use); water, 0.05–0.10 wt% max.; acidity (as acrylic acid), 0.009 wt% max.; hydroquinone (inhibitor), 40–160 ppm; monomethyl ether of hydroquinone (inhibitor), 10–220 ppm (Union Carbide Corp., 1982; Hoechst Celanese Corp., 1988).

1.1.5 *Analysis*

Methods for sampling and analysing air have been developed for vapours of acrylate monomers, including 2-ethylhexyl acrylate. The acrylate monomer vapour is adsorbed on activated silica gel or charcoal, desorbed in acetone or carbon disulfide and analysed by gas chromatography with flame ionization detection (Bosserman & Ketcham, 1980; Samimi & Falbo, 1982). The limit of sensitivity is 0.01 ppm [0.075 mg/m^3] (Bosserman & Ketcham, 1980). Steichen (1976) described a head-space method for the determination of residual 2-ethylhexyl acrylate monomer in the polymer by gas chromatography–flame ionization detection, with a limit of detection of 5 ppm [mg/kg].

1.2 Production and use

1.2.1 *Production*

Direct, acid-catalysed esterification of acrylic acid with 2-ethylhexanol is the principal method for the manufacture of 2-ethylhexyl acrylate. The commonest catalysts are sulfuric and *para*-toluenesulfonic acid and sulfonic acid functional cation-exchange resins. The monomethyl ether of hydroquinone is added as a polymerization inhibitor, and the esters are used in this form in most industrial applications (Ohara *et al.*, 1985; Bauer, 1991; Tyler, 1993).

Estimated production volumes of 2-ethylhexyl acrylate in the USA in 1980, 1985, 1990 and 1991 were 31, 36, 53 and 48 thousand tonnes, respectively (Mannsville Chemical Products Corp., 1984; US International Trade Commission, 1986, 1991, 1993). In 1990, about 50 thousand tonnes were manufactured in Germany (Beratergremium für umweltrelevante Altstoffe, 1993).

1.2.2 *Use*

Acrylic esters are used in the production of polymers and copolymers with a wide range of applications. As a plasticizing co-monomer, 2-ethylhexyl acrylate is used in the production

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[a]Calculated from: mg/m^3 = (relative molecular mass/24.45) × ppm, assuming normal temperature (25 °C) and pressure (101.3 kPa)

of resins for pressure-sensitive adhesives, latex, paints, textile and leather finishes and coatings for paper. 2-Ethylhexyl acrylate can also be used as a co-monomer in solution polymers for industrial metal finishing (Mannsville Chemical Products Corp., 1984; Ohara *et al.*, 1985; Tyler, 1993; Hoechst Celanese Corp., undated).

The major current use of 2-ethylhexyl acrylate is in acrylic pressure-sensitive adhesives, of which it is a major component. The typical composition of an adhesive for general-purpose tape is 75% 2-ethylhexyl acrylate, 20% vinyl acetate, 4% acrylic acid and 1% *N*-methylolacrylamide (see monograph, p. 435) (Temin, 1990).

2-Ethylhexyl acrylate is also used in ultraviolet-curable coatings without solvents, which provide a glossy, abrasion-resistant finish, e.g. on book covers and record albums. A typical ultraviolet-cured formulation might include 20% trimethylpropane triacrylate, 70% acrylated polyurethane oligomer, 10% 2-ethylhexyl acrylate diluent monomer and small amounts of photoinitiator. A liquid coating or ink is spread on the surface of the substrate, and the coating is exposed to ultraviolet light for less than 1 sec and is completely cured (Mannsville Chemical Products Corp., 1984).

The estimated use patterns of acrylic esters, including 2-ethylhexyl acrylate, in Japan, western Europe and the USA are presented in Table 1 (Ohara *et al.*, 1985).

Table 1. Estimated distribution of uses of acrylic esters (% of total)

Use	Japan	Western Europe	USA
Surface coatings	34	35	42
Textiles	16	18	23
Acrylic fibres	14	7	6
Adhesives	20	15	5
Other	16	25	24

From Ohara *et al.* (1985)

1.3 Occurrence

1.3.1 *Natural occurrence*

2-Ethylhexyl acrylate is not known to occur as a natural product.

1.3.2 *Occupational exposure*

The National Occupational Exposure Survey conducted by the National Institute for Occupational Safety and Health between 1981 and 1983 indicated that 11 300 US employees were potentially exposed to 2-ethylhexyl acrylate (US National Institute for Occupational Safety and Health, 1993). Of this number, 53% were estimated to be exposed to pure 2-ethylhexyl acrylate and 47% to products containing it. The estimate is based on a survey of US companies and did not involve measurements of actual exposures.

Few data have been reported on occupational exposure to 2-ethylhexyl acrylate. Exposures of workers to styrene (see p. 239) and several acrylates, including 2-ethylhexyl acrylate, and area concentrations were monitored in a US facility where acrylic ester–styrene copolymers were produced. The concentrations of 2-ethylhexyl acrylate in 11 personal samples collected for various times at a process reactor which had an opening hatch for addition of starting products (reactor A) ranged from not detectable to 2 ppb [15 μg/m^3] (mean, 0.4 ppb [3 μg/m^3]); nine personal samples taken at a similar reactor contained no detectable concentration, but 13 personal samples collected on workers tending a completely closed reactor contained no detectable compound to 5 ppb [38 μg/m^3] (mean, 1 ppb [7.5 μg/m^3]). No detectable concentrations were found in six personal samples taken from workers at a closed polymer flake continuous reactor, and those in 11 personal samples collected at the unloading docks ranged from not detectable to 5 ppb [38 μg/m^3] (mean, 2 ppb [15 μg/m^3]). In all cases, exposures to ethyl acrylate, *n*-butyl acrylate, methyl methacrylate (see monograph, p. 445), styrene (see monograph, p. 233) and *alpha*-methylstyrene exceeded the levels of 2-ethylhexyl acrylate. All 49 area samples taken in the same production areas, except reactor A, had no detectable levels of 2-ethylhexyl acrylate, but eight area samples taken at reactor A had levels ranging from not detectable to 161 ppb [1.2 mg/m^3] (mean, 30 ppb [226 μg/m^3]) (Samimi & Falbo, 1982).

Data on exposure of workers during manufacture of 2-ethylhexyl acrylate in four US plants are summarized in Table 2. Since 2-ethylhexyl acrylate is used primarily as an intermediate in closed process reactors, the concentrations are generally expected to be low. Exposure by inhalation would also be expected to be low in view of the vapour pressure of this compound (19 Pa); however, dermal exposures may occur during spills or leaks (Björkner *et al.*, 1980).

1.3.3 *Water*

2-Ethylhexyl acrylate was detected at concentrations ranging from 0.6 to 11 ppb (μg/L) (mean, 4 ppb) in the effluent from the last stage of an on-site waste-treatment facility which received water from a large petrochemical plant on the US coast of the Gulf of Mexico. The influent untreated wastewater contained 0.55–5.6 ppm (mg/L) (mean, 2.0 ppm) (Berglund & Whipple, 1987).

1.4 Regulations and guidelines

Occupational standards or guidelines have not been established for 2-ethylhexyl acrylate (ILO, 1993; American Conference of Governmental Industrial Hygienists, 1993; UNEP, 1993). Russia has a short-term exposure limit of 1 mg/m^3 (ILO, 1993). Union Carbide, a major supplier of 2-ethylhexyl acrylate, has adopted a threshold limit value of 5 ppm [38 mg/m^3] for its internal operations (Samini & Falbo, 1982).

The US Food and Drug Administration (1993) has established regulations for the use of monomers, polymers and copolymers of 2-ethylhexyl acrylate in products in contact with food.

Table 2. Concentrations of 2-ethylhexyl acrylate to which workers are exposed during manufacture

Operation	No. of samples	Concentration in personal air samples			
		Geometric mean		Range	
		ppb	mg/m^3	ppb	mg/m^3
Full-shift time-weighted average (> 336 min)					
Operations A	12[a]	500	3.8	100–900	0.75–6.8
Operations B	60	50	0.4	1–1100	0.01–8.3
Quality assurance	9	30	0.2	< 10–120	< 0.1–0.9
Maintenance	63	140	1.1	3–460	0.02–3.5
Loading and unloading	20	160	1.2	100–600	0.75–4.5
Short-term (< 15 min)					
Operations A	1	500	3.8	500	3.8
Operations B	5[a]	220	1.7	130–280	1.0–2.1
Maintenance and use	1	100	0.75	100	0.75
Loading and unloading	1	< 100	< 0.75	< 100	< 0.75

From Tyler (1993)
[a]Area samples

2. Studies of Cancer in Humans

No data were available to the Working Group.

3. Studies of Cancer in Experimental Animals

3.1 Skin application

Mouse: A group of 40 male C3H/HeJ mice, 7–10 weeks of age, received skin applications of about 20 mg/mouse 75% 2-ethylhexyl acrylate (purity, 99%) dissolved in acetone three times per week for life. Two groups of 40 mice were given applications of acetone alone (negative control), and one group of 40 mice was given about 0.03 mg/mouse 0.2% 3-methylcholanthrene (positive control) by the same dose schedule. The numbers of mice alive in the two acetone control groups and in the 2-ethylhexyl acrylate-treated group were 33, 19 and 30 at one year and 22, 13 and 15 at 1.5 years, respectively. Six skin tumours (two squamous-cell carcinomas and four papillomas) developed in the group treated with 2-ethylhexyl acrylate but in neither of the acetone control groups [$p = 0.001$, Fisher exact test]; 35/40 animals in the positive control group developed skin tumours, 34 of which were malignant (DePass, 1982; DePass *et al.*, 1985).

Groups of 80 male C3H/HeJ mice, six weeks of age, received skin applications of 2.5, 21, 43 or 86.5% (w/w) 2-ethylhexyl acrylate (purity, 99.5%) in 25 μl acetone three times per week

for life, except that treatment of the group given 43% was stopped at 24 weeks (stop test). Two groups of 80 mice served as untreated or acetone-treated controls. The mean body weights were slightly greater in the treated groups than in the controls. No treatment-related effect on survival was observed: median survival was 97–102 weeks in treated mice and 105–108 weeks in controls. Skin tumours were seen in the groups treated with 21 and 86.5%: papillomas in 5 and 10 mice, respectively [$p < 0.001$ when compared with none in control groups]; squamous-cell carcinomas in 20 [$p < 0.001$, Fisher exact test] and 16 mice [$p < 0.001$ when compared with none in control groups, Fisher exact test]; malignant melanomas in 7 [$p = 0.001$] and 9 mice [$p < 0.0015$ when compared with none in control groups, Fisher exact test]. One basal-cell carcinoma and five fibrosarcomas were also seen in the 21% group and one haemangioma in the 86.5% group. No skin tumour occurred in the group given 2.5% or in the stop-test group. Scaling and scabbing were observed in all treated groups and persisted throughout the treatment period. Regression of the lesions was reported to have occurred within seven weeks after cessation of treatment of the group with 43%. Hyperkeratosis and hyperplasia had occurred in all groups, however, by the end of treatment (Wenzel-Hartung *et al.*, 1989).

Groups of 80 male NMRI mice, seven weeks of age, received skin applications of 0, 21.5, 43 or 85% (w/w) 2-ethylhexyl acrylate (purity, > 99.7%) in 25 µl acetone three times per week on clipped dorsal skin. A positive control group of 80 mice received 0.015% benzo[*a*]pyrene in 25 µl acetone by the same dose schedule. After seven months of treatment, each group, including the positive control group, was divided into two subgroups. One continued to receive the original treatment for the remainder of the two-year study period; in the second subgroup, treatment was discontinued, and, after two months, the mice were treated with 5 µg/animal 12-*O*-tetradecanoylphorbol 13-acetate (TPA) in 0.1 ml acetone twice a week for 20 weeks and then observed for the remainder (about nine months) of the two-year study. Body weights and survival were not affected by treatment with 2-ethylhexyl acrylate, with or without TPA. At termination of the study, 13–31% of the mice treated with 2-ethylhexyl acrylate and 17–19% of the acetone controls with or without TPA treatment were still alive. No skin tumour was seen in the groups treated with 2-ethylhexyl acrylate without TPA or in the acetone controls. One squamous-cell papilloma of the skin was seen in each of the groups painted with 21.5, 43 and 85% 2-ethylhexyl acrylate and TPA. Squamous-cell carcinomas were observed only in the benzo[*a*]pyrene-treated mice treated with and without TPA. Hyperkeratosis and hyperplasia occurred in all treated groups (Mellert *et al.*, 1994).

4. Other Data Relevant for an Evaluation of Carcinogenicity and Its Mechanisms

4.1 Absorption, distribution, metabolism and excretion

4.1.1 *Humans*

No data were available to the Working Group.

4.1.2 *Experimental systems*

Elimination of total radioactivity from tissues was measured in two studies after administration of 2-ethylhexyl [2,3-^{14}C]acrylate to groups of Wistar rats (Gut *et al.*, 1988; Sapota, 1988). In the study of Gut *et al.* (1988), doses of 10 mg/kg bw were administered either intraperitoneally or intravenously. One hour after dosing, only small amounts of metabolized compound were found in blood and other tissues; the tissues with the highest percentages of total radioactivity after intravenous administration were kidney (25.5%), liver (12.8%), brain (7.7%), thymus (7.4%), spleen (6.4%) and blood (2.6%). Elimination of radioactivity from blood was bi-exponential and was dependent on route of administration and age (weight) of the rats. The half-lives for the initial phase after intravenous and intraperitoneal administration were 30 min and 60 min in four-month-old (265–295 g) rats and 115 min and 130 min in seven-month-old (450–500 g) rats, respectively. The corresponding half-lives for the terminal phase were 5 and 6 h and 14 and 14 h. Elimination from other tissues followed a pattern similar to that of blood. Less than 0.01% of an administered dose was excreted in the faeces and 13.5% of an intravenous dose and 7.2% of an intraperitoneal dose were excreted in the urine within 24 h; about 2% of an intraperitoneal dose of 1 mmol/kg bw was excreted as thioethers. About 50% of the administered dose was expired, mostly as carbon dioxide, during the first 24 h after dosing, when about two-thirds of the radioactivity could be accounted for.

In the study of Sapota (1988), doses of 100 mg/kg bw were administered either orally or by intraperitoneal injection. Direct comparison with the results of Gut *et al.* (1988) is not possible, but about 75% of the administered radioactivity was found within 24 h in exhaled air after intraperitoneal treatment and 50% after oral treatment. Elimination from erythrocytes was biphasic, whereas elimination from plasma was monophasic and had a half-life of about 22 h. [The difference of the results from those of Gut *et al.* may be due to use of a higher dose, which would have saturated elimination pathways. Studies of distribution and elimination of total radioactivity are generally of limited relevance for toxicological evaluations.]

2-Ethylhexyl acrylate is believed to undergo carboxylesterase-catalysed metabolism, like other acrylate esters (Miller *et al.*, 1981). It is excreted in the urine both as *N*-acetyl-*S*-(2-carboxyethyl)cysteine and as *N*-acetyl-*S*-2-(2-ethylhexyloxycarbonyl)ethylcysteine (Kopecký *et al.*, 1985). Two unidentified metabolites were detected in the bile of rats (Cikrt *et al.*, 1986). In rats exposed by inhalation, the percentage of a dose of the acrylate excreted in urine as thioethers over 24 h was dose dependent and decreased from 8.0 to 3.0% as the 6-h exposure concentration in air increased from 250 to 1000 mg/m^3, indicating saturable metabolism along this pathway. A decrease in the number of non-protein –SH groups was also observed in the blood and liver of these animals (Vodička *et al.*, 1990).

2-Ethylhexyl acrylate significantly increased bile flow (Cikrt *et al.*, 1986) and blood glucose level (Vodička *et al.*, 1990) in rats.

2-Ethylhexyl acrylate is an α,β-unsaturated carbonyl compound which reacts in Michael-type additions with nucleophiles (Tyler, 1993). No study is available, however, on the identity of putative adducts formed by 2-ethylhexyl acrylate or any of its metabolites with proteins or DNA *in vivo*.

4.2 Toxic effects

The toxicology of 2-ethylhexyl acrylate has been reviewed (Beratergremium für umweltrelevante Altstoffe, 1993).

4.2.1 *Humans*

No data were available to the Working Group.

4.2.2 *Experimental systems*

Sensitization was observed when guinea-pigs were treated with 2-ethylhexyl acrylate in Freund's complete adjuvant (Waegemaekers & van der Walle, 1983).

4.3 Reproductive and prenatal effects

No data were available to the Working Group.

4.4 Genetic and related effects

4.4.1 *Humans*

No data were available to the Working Group.

4.4.2 *Experimental systems* (see also Table 3 and Appendices 1 and 2)

2-Ethylhexylacrylate did not induce mutation in *Salmonella typhimurium*. Equivocal results for mutation at the *tk* and *hprt* loci were seen in Chinese hamster ovary and L5178Y mouse lymphoma cells *in vitro*.

It is unclear whether 2-ethylhexylacrylate is clastogenic. Equivocal results were obtained for small colony formation and chromosomal aberrations in L5178Y mouse lymphoma cells. It did not induce micronuclei in these cells.

5. Summary of Data Reported and Evaluation

5.1 Exposure data

2-Ethylhexyl acrylate is produced by acid-catalysed esterification of acrylic acid with 2-ethylhexanol. Its major uses are in pressure-sensitive adhesives, in resins for latex paints and paper coatings and in the finishing of textiles. Occupational exposure to 2-ethylhexyl acrylate has been reported during its production and use.

5.2 Human carcinogenicity data

No data were available to the Working Group.

Table 3. Genetic and related effects of 2-ethylhexyl acrylate

Test system	Result[a]		Dose[b] (LED/ HID)	Reference
	Without exogenous metabolic system	With exogenous metabolic system		
SA0, *Salmonella typhimurium* TA100, reverse mutation	–	–	5000	Zeiger *et al.* (1985)
SA5, *Salmonella typhimurium* TA1535, reverse mutation	–	–	5000	Zeiger *et al.* (1985)
SA7, *Salmonella typhimurium* TA1537, reverse mutation	–	–	5000	Zeiger *et al.* (1985)
SA9, *Salmonella typhimurium* TA98, reverse mutation	–	–	5000	Zeiger *et al.* (1985)
GCO, Gene mutation, Chinese hamster ovary cells *in vitro*, *hprt* locus (suspension assay)	–	0	26	Moore *et al.* (1991)
GCO, Gene mutation, Chinese hamster ovary cells *in vitro*, *hprt* locus	?	0	80	Moore *et al.* (1991)
G5T, Gene mutation, mouse lymphoma L5178Y cells, *tk* locus	?	0	34	Dearfield *et al.* (1989)
MIA, Micronucleus formation, L5178Y mouse lymphoma cells	–	0	34	Dearfield *et al.* (1989)
CIM, Chromosome aberrations, L5178Y mouse lymphoma cells	?	0	34	Dearfield *et al.* (1989)

[a] +, positive; –, negative; 0, not tested; ?, inconclusive (variable response within several experiments within an adequate study)
[b] In-vitro tests, μg/ml; in-vivo tests, mg/kg bw

5.3 Animal carcinogenicity data

2-Ethylhexyl acrylate was tested by skin application in three experiments in mice. It increased the incidence of squamous-cell carcinomas of the skin in two experiments and of malignant melanomas in one experiment. In the third experiment, in a different strain of mice, 2-ethylhexyl acrylate did not increase skin tumour incidence, with or without subsequent application of 12-*O*-tetradecanoylphorbol 13-acetate.

5.4 Other relevant data

2-Ethylhexylacrylate is rapidly metabolized in rats; a small proportion is exhaled as carbon dioxide within 24 h, and a small proportion is excreted as thioethers in urine. No data were available on the genetic and related effects of 2-ethylhexylacrylate in humans. There is very little evidence for or against its genotoxicity in experimental systems.

5.5 Evaluation[1]

There is *inadequate evidence* in humans for the carcinogenicity of ethylhexyl acrylate.

There is *limited evidence* in experimental animals for the carcinogenicity of ethylhexyl acrylate.

Overall evaluation

Ethylhexyl acrylate *is not classifiable as to its carcinogenicity to humans (Group 3).*

6. References

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[1]For definition of the italicized terms, see Preamble, pp. 27–30.

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