Phenoxyethyl acrylate

48145-04-6

OVERVIEW

Prepared for the National Cancer Institute (NCI) for consideration by the Chemical Selection Working Group (CSWG) by Technical Resources International, Inc. under contract no. N02-07007.

Phenoxyethyl acrylate came to the attention of the National Cancer Institute (NCI) Division of Cancer Biology as the result of a review of high production chemicals in commerce that do not meet the criteria for inclusion in the United States (U.S.) Environmental Protection Agency (EPA) HPV Challenge Program. This compound has applications in the adhesives, graphic arts, composites, inks, ultraviolet (UV)-curing coatings, and photoresists electronics industries. Occupations that have documented exposure to phenoxyethyl acrylate include janitors, cleaners, and printing machine operators who work with rubber and miscellaneous plastics products.

The available information on phenoxyethyl acrylate is insufficient to establish a toxicological profile for this chemical. Aside from a small number of acute studies, no data on the toxicity of this chemical was found in the available literature. As a result, phenoxyethyl acrylate was submitted to the NTP Genotoxicity Program by the Chemical Selection Planning Group (CSPG) in July 2004. Concerns over harmful breakdown products of phenoxyethyl acrylate were raised at this meeting.

INPUT FROM GOVERNMENT AGENCIES/INDUSTRY

Dr. Boris Ionin from the Department of Bacterial Diseases of the Walter Reed Army Institute of Research provided a translation for the Bitkina *et al.* reference.

NOMINATION OF PHENOXYETHYL ACRYLATE TO THE NTP

Based on a review of the available literature and the recommendations of the Chemical Selection Working Group (CSWG) on December 15, 2004, NCI nominates this chemical for testing by the National Toxicology Program (NTP) and forwards the following information:

- The attached Summary of Data for Chemical Selection
- Copies of references cited in the Summary of Data for Chemical Selection
- CSWG recommendations to:

(1) Characterize the toxicity of phenoxyethyl acrylate in a 90 day study.

(2) Conduct metabolic and disposition studies to identify breakdown products that would differentiate between epoxidation and hydroxylation as the primary metabolic process.

PRIORITY

The CSWG suggested that the recommended testing be conducted with moderate to high priority.

SUMMARY OF DATA FOR CHEMICAL SELECTION

CHEMICAL IDENTIFICATION

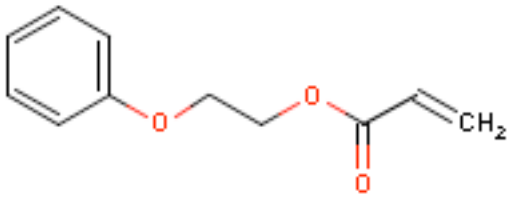
CAS Registry No: 48145-04-6

Chemical Abstracts Service Name: 2-Propenoic acid, 2-phenoxyethyl ester (9CI)

Synonyms and Trade Names: Phenoxyethyl acrylate; EINECS 256-360-6; ethylene glycol phenyl ether acrylate; 2-phenoxyethanol acrylate; phenylcellusolve acrylate; (ChemFinder, 2004; ChemIDplus, 2004)

Structural Class: Acrylate; glycol ether

Structure, Molecular Formula, and Molecular Weight:



$C_{11}H_{12}O_3$ Mol. wt.: 192.2

Chemical and Physical Properties:

Description: Colorless to yellowish liquid with a pungent odor (BASF Aktiengesellschaft, 2002)

Melting point: -36 ºC (BASF Aktiengesellschaft, 2002)

Boiling point: 287 ºC (Bitkina *et al.*, 1994); 111 ºC at 2.7 mbar (BASF Aktiengesellschaft, 2002)

Solubility: Practically insoluble in water; very soluble in acetone, ether, chloroform, and vegetable oils (Bitkina *et al.*, 1994; Lide, 2004)

Density/Specific Gravity:: 1.09 at 25 ºC (Lide, 2004)

Flash point: 113 ºC closed cup (Sigma Aldrich MSDS, 2004)

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Reactivity:	Can polymerize if shelf life or storage temperature are greatly exceeded. Reacts with peroxides and other radical components. Heat develops during polymerization. Ignitable air mixtures can form when the product is heated above the flash point or when sprayed (BASF Aktiengesellschaft, 2002)

Technical Products and Impurities:

Phenoxyethyl acrylate stabilized with 100 ppm hydroquinone (HQ) is available from Sigma Aldrich although the purity was not specified (Sigma Aldrich, 2004).

EXPOSURE INFORMATION

Production and Producers:

Manufacturing Process. Two principal processes are used for the manufacture of monomeric acrylic esters, the semicatalytic Reppe process and the propylene oxidation process. The newer propylene oxidation process is preferred because of economy and safety. In this process acrolein is first formed by the catalytic oxidation of propylene vapor at high temperature in the presence of steam. The acrolein is then oxidized to acrylic acid. Both one-step and two-step oxidation processes are known. A number of catalyst systems may be employed; most use a molybdenum compound as the main component. The acrylic acid is esterified with alcohol to the desired acrylic ester in a separate process. In normal practice, inhibitors such as hydroquinone (HQ) or the momomethyl ether of hydroquinone (MEHQ) are added to stabilize acrylic monomers during shipment and storage. Removal of the stabilizers before use is not normally required. Commercially, acrylic monomers are shipped in bulk quantities, tank cars, or tank trucks (Novak, 1991).

Producers and Importers. Six U.S. producers or distributors of phenoxyethyl acrylate are listed by Chemical Sources International (2004). According to recent issues of chemical directories, phenoxyethyl acrylate is manufactured and/or distributed by Albemarle Corporation; Ciba Specialty Chemicals, Water Treatments Div.; Jarchem Industries Inc.; Lancaster; Monomer-Polymer & Dajac Labs, Inc.; San Esters Corp.; Sartomer Company; and TCI (ChemACX, 2004; Chemcyclopedia, 2004; Chemical Information Services, 2004; Chemical Week Associates, 2004; Tilton, 2003).

Several corporations produce bulk quantities of phenoxyethyl acrylate under trade names identified with specific applications. These include LAROMER® POEA produced by BASF AG, PHOTOMER 4035 produced by Cognis, AGEFLEX™ PEA by Ciba Specialty Chemicals, SR 339 by Sartomer, and EBECRYL 114 by UCB Chemicals (BASF Aktiengesellschaft, 2002; Ciba Specialty Chemicals, 2002; Cognis Corporation, 2003; Sartomer, 1998; UCB Chemicals, 2004).

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Production/Import Level. Phenoxyethyl acrylate is listed in the EPA Toxic Substances Control Act (TSCA) Inventory (ChemIDplus, 2004). The annual production ranges, supplied to EPA under the Inventory Update Rule (IUR) every four years, indicate increased production of phenoxyethyl acrylate beginning in 1998. Information from 1986 to 2002 IURs is listed in Table 1.

Table 1. Production Levels of Phenoxyethyl Acrylate

Year	Production Range (lbs.)
1986	10,000 - 500,000
1990	> 500,000 - 1,000,000
1994	10,000 - 500,000
1998	> 1,000,000 - 10,000,000
2002	> 1,000,000 - 10,000,000

Source: EPA (2004b)

Phenoxyethyl acrylate is also an HPV chemical in Europe where quantities in excess of 1,000 metric tons were produced or imported between 1990 and 1994 (European Commission, 2000a; ESIS, 2004).

Use Pattern:

Phenoxyethyl acrylate monomer is a hydrophobic monomer that imparts solubility and wetting properties for adhesion and pigment carrying. Phenoxyethyl acrylate polymers offer excellent abrasion resistance; resistance to polar solvents, such as water, acids, and alcohols; and weatherability (Jarchem Industries, Inc., 2003; Sartomer Company, Inc., 1996, 1998 & 2004a).

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Phenoxyethyl acrylate monomer has applications in high energy electron radiation-curable coatings where it serves as an analogue of the solvent in conventional paints. In addition to its use as a thinner, the acrylic groups allow phenoxyethyl acrylate monomer to also serve as the crosslinking component in UV- and electron beam (EB)-curing systems. The phenoxyethyl acrylate becomes part of the polymer structure during curing (BASF Aktiengesellschaft, 2004; McGinniss, 1996).

The polymerizable acrylic groups in phenoxyethyl acrylate enable this chemical to form copolymers of acrylic or methacrylic acids and their salts, amides, esters, vinyl acetate, and styrene. These properties make phenoxyethyl acrylate an important feedstock for synthesis in the chemical industry.

One of the main uses of phenoxyethyl acrylate is in coatings on glass, metal, paper, plastic, wood, and PVC floor coatings. The composition of phenoxyethyl acrylate in coatings can be substantial; for coating polyethylene terephthalate, 12.2% by weight was reportedly used (Univar, 2004).

Major manufacturers tend to market trade-name monomers to targeted audiences.

- Laromer® POEA is marketed as a reactive thinner for radiation-curable coatings, as a feedstock for syntheses, and for manufacturing polymers (BASF Akeiengesellschaft, 2004).
- Suggested applications for Sartomer SR-339 include adhesives; photoresists electronics; glass, optical, and metal coating; paper, plastic, and PVC floor coatings; and flexo, gravure, and screen inks (Sartomer Company, 1998).
- AGEFLEX™ PEA has applications for gloss in flexo, gravure, and screen inks; can be used in UV and EB coatings for optical fiber; and is employed in ophthalmic products such as contact lenses (Ciba Specialty Chemicals, 2002).
- PHOTOMER® 4035 has applications in adhesives, graphic arts, and composites (Cognis Corporation, 2003).

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- EBECRYL 114 is one of a group of diluting acrylates marketed for UV/EB curing (UCB Chemicals, 2004).

Phenoxyethyl acrylate applications in ophthalmic products and as a coating for optical fibers are attributed to its high index of refraction (Ciba Specialty Chemicals, 2002). This chemical has also been reported as a component in carbonless copy paper (CarbonlessCopyPaper.com, 2004).

Phenoxyethyl acrylate and 2-phenoxyethyl acrylate are cited in 1,047 and 460 U.S. patents from 1976 to the present, respectively (United States Patent and Trademark Office, 2004).

Human Exposure:

Occupational Exposure. The primary source of human exposure to phenoxyethyl acrylate monomer is the workplace, both in the manufacture of this chemical and in its many applications.

Due to the low volatility of phenoxyethyl acrylate, human exposure via inhalation at room temperature is limited. However, aerosols or vapors may be generated at elevated processing temperatures, resulting in much higher airborne concentrations (Sartomer Company Inc., 2004b).

Extensive, prolonged, or repeated exposure to phenoxyethyl acrylate monomer can result in significant dermal absorption (Sartomer Company Inc., 2004b).

Due to the concern about dermal absorption, industry has conducted a glove permeation study to evaluate the protection provided from UV/EB-curing acrylates to workers wearing nitrile gloves. Nitrile gloves of varying thickness typically used in work situations were assessed based on exposure duration: thin gloves for brief contact with the acrylates (less than 1 hour), medium gloves for longer exposure, e.g. from opening a drum (up to 4 hours), and thick gloves for longest exposure, e.g., from cleaning print or coating equipment

contaminated with acrylates (8 hours). Thin and medium thickness gloves did not prevent permeation of phenoxyethyl acrylate during the periods of their intended use. Although thick gloves were not tested, the authors estimated that phenoxyethyl acrylate would penetrate such gloves in less than 2 hours (Zwanenburg, 2000).

Phenoxyethyl acrylate monomer may be used as a solvent, dispersion, or emulsion substance to produce polymers for paints and varnishes.

For methyl methacrylate, similar products are produced by reactions carried out in semiautomated batch reactors performed within a closed system. Exposure may occur during handling, filling, sampling operations and waste treatment. Likewise, preparation of reactive resins used in floor coatings and adhesives would result in possible exposure during sampling and analysis, filling and drumming, and during cleaning, maintenance, and repair work. Preparation of polymeric resins at the site of application would result in possible exposure to the construction laborer applying the coating (European Commission, 2002b).

It would be expected that similar exposures would occur from the production and uses of phenoxyethyl acrylate.

It appears that a considerable quantity of phenoxyethyl acrylate is further processed by customers. As noted for methyl methacrylate, it can be assumed that further processing is performed not only in the large-scale chemical industry but also in companies with lower levels of protection. In these areas, it must be presumed that the substance may be handled in open systems during certain tasks, e.g. metering and filling activities or application works, and that suitable technical measures (local exhaust ventilation, personal protective equipment, and gloves) may not be used (European Commission, 2002b).

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The National Occupational Exposure Survey (NOES), which was conducted by the National Institute for Occupational Safety and Health (NIOSH) between 1981 and 1983, estimated that 724 workers, including 580 females, in 48 facilities representing 1 industry were potentially exposed to phenoxyethyl acrylate in the workplace (BiblioLine, 1998). The NOES database does not contain information on the frequency, level, or duration of exposure to workers of any listed chemical. This information also does not reflect recent increases in the use of phenoxyethyl acrylate.

Estimates from the NOES suggest that individuals working as janitors, cleaners, or printing machine operators have more potential exposure to phenoxyethyl acrylate than other occupations (NIOSH, 2004). However, the greatest exposure would be expected to occur in workers involved in the manufacture of phenoxyethyl acrylate and in workers where phenoxyethyl acrylate monomer is used in downstream products.

Environmental Exposure. Human exposure to phenoxyethyl acrylate in the environment may occur from waste streams produced from the manufacturing, use, and disposal of phenoxyethyl acrylate. Since phenoxyethyl acrylate monomer is not very stable, this chemical should not be a persistent pollutant.

Consumer Exposure. Consumers may be exposed to small amounts of unreacted phenoxyethyl acrylate monomer leached from products such as carbonless copy paper, contact lenses, and inks due to incomplete reaction.

Based on the low migration rate, the European Union has estimated that consumer methyl methacrylate exposure by skin contact with polymethyl methacrylate or oral intake from use of polymethyl methacrylate articles would be negligible (European Commission, 2002b). Likewise, it is projected that consumer exposure to phenoxyethyl acrylate monomer from skin contact with polymeric forms would be negligible.

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Environmental Occurrence:

Phenoxyethyl acrylate may be released into various waste streams during manufacture and use. This chemical is regarded by industry as toxic to aquatic organisms, and it may cause long-term adverse effects in the aquatic environment (BASF Aktiengesellschaft, 2002).

Regulatory Status:

No standards or guidelines have been set by NIOSH or OSHA for occupational exposure to or workplace allowable levels of phenoxyethyl acrylate. Phenoxyethyl acrylate was not on the American Conference of Governmental Industrial Hygienists (ACGIH) list of compounds for which recommendations for a Threshold Limit Value (TLV) or Biological Exposure Index (BEI) are made.

According to one manufacturer, monomers properly inhibited with HQ or MEHQ are generally not RCRA hazardous wastes. However, it is the responsibility of the waste generator to determine if the product meets the criteria of a hazardous waste at the time of disposal (Sartomer, 2004a).

Based on cancer concerns, new chemicals in the acrylates category were regulated by EPA under Section 5 of TSCA. Following discussions with EPA staff, industry members of the Specialty Acrylates Manufacturers (SAM) offered to conduct a voluntary testing program on 13 existing acrylates in exchange for concessions by EPA in regulating new acrylates under Section 5. As the result of the negotiations, EPA agreed not to require a cancer warning on product labels for new acrylates regulated by EPA; however, cancer warnings would still appear on the Material Safety Data Sheets for these chemicals. For its part, SAM agreed in 1990 to conduct a voluntary testing program involving physical/chemical properties and metabolism/pharmacokinetics testing on the 13 existing acrylates and cancer bioassays on two of those chemicals. SAM's voluntary testing program was completed in September 1995 (EPA, 2004a). Phenoxyethyl acrylate was not one of the 13 acrylates tested by SAM.

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TOXICOLOGICAL INFORMATION

Human Data:

No epidemiological studies or case reports investigating exposure to phenoxyethyl acrylate and cancer risk in humans were identified in the available literature.

A sensitization study of phenoxyethyl acrylate was submitted by AT&T as a TSCA Section 8(e) document. This study consisted of dermal patch testing of AT&T employees who had exhibited dermal reactions after working with various fiber coating formulations containing phenoxyethyl acrylate. Eight out of 47 (17%) individuals responded to phenoxyethyl acrylate at 0.1% and 24 out of 43 (56%) responded to phenoxyethyl acrylate at 1.0% (AT&T, 1992).

Animal Data:

Acute Studies. LD_{50} values for phenoxyethyl acrylate are listed in Table 2.

Table 2. Acute Toxicity Values for Phenoxyethyl Acrylate

Species	Route of Administration	LD_{50} (mg/kg)
rat	oral	~ 5,000
rat, male	oral	5,500
rat, female	oral	5,000
mouse	oral	4,500
rabbit	skin	~ 2,800

Source: Biblioline, 1998; Bitkina *et al*., 1994

Following acute intoxication with phenoxyethyl acrylate from intragastric or intraperitoneal administration, laboratory rodents developed lateral recumbency within 2-3 hours and death

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at 3 days post-exposure. Inhalation exposure at the saturating concentration did not induce any symptoms of intoxication (Bitkina *et al*., 1994).

The irritation and sensitization potential of phenoxyethyl acrylate was studied using rabbits and guinea pigs. Application to the mucous membrane of the rabbit eye resulted in moderate irritation. When 500 mg was applied to rabbit skin, phenoxyethyl acrylate produced a mild irritation. No pathological effect was observed in guinea pigs given a single application of phenoxyethyl acrylate to the skin, although repeated (4-5) applications led to the development of acute dermatitis (Biblioline, 1998; Bitkina *et al*., 1994).

Repeat Dose Studies. Daily intragastric administration of phenoxyethyl acrylate to laboratory animals for 30 days at one tenth the LD_{50} produced adverse effects, including a decrease in weight gain, increase in urine protein content, and decreases in hemoglobin level and erythrocyte count. An increase in alkaline phosphatase activity was also reported. Dystrophic changes in the cells of the liver and of the proximal tubule epithelium of the kidneys were observed, and hyperplasia with hyperkeratosis was noted in the lymphoid follicles of the spleen and in the pregastric epithelium. These effects persisted after 2 weeks of recovery and had not resolved completely at 2 months post-exposure. Inflammatory gastroenterocolitis was a chronic condition in treated animals with increased mitotic activity of the gastric and intestinal epithelium. Important details of the study protocol, such as the species, sex, or number of animals, were not reported (Bitkina *et al*., 1994).

Chronic/Carcinogenicity Studies: No 2-year carcinogenicity studies of phenoxyethyl acrylate in animals were identified in the available literature.

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Short-term Tests:

No *in vitro* or *in vivo* studies evaluating phenoxyethyl acrylate for mutagenic effects were found in the available literature. Phenoxyethyl acrylate was submitted by the NCI for testing by the NTP Genotoxicity Program in October 2004.

Metabolism:

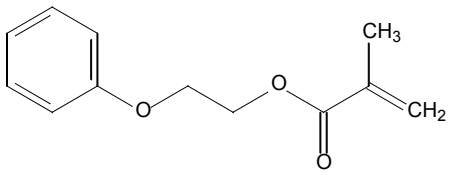
No studies on the metabolism of phenoxyethyl acrylate were found in the available literature. During the evaluation of this chemical, the CSPG proposed that an esterase could cleave the molecule at its ether linkage and might release acrylic acid.

Other Biological Effects:

No other relevant toxicological information for phenoxyethyl acrylate was found in the available literature.

Structure/Activity Relationships:

Based on a search of chemicals in the ChemID database with 80% or greater structural similarity to phenoxyethyl acrylate, two chemicals were identified, phenoxyethyl propionate and 2-phenoxyethyl methacrylate.



Phenoxyethyl methacrylate (10595-06-9)

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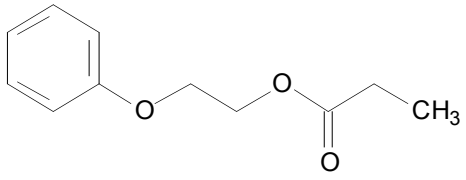

2-Phenoxyethyl propionate (23495-12-7)

No information on the carcinogenicity or genotoxicity of either chemical was identified in a search of the available literature.

The TOXNET database, CCRIS was also compared against the list of acrylates and methacrylates removed from EPA's Master Testing List (MTL) following industry testing. The following MTL chemicals were not in CCRIS: phenyl acrylate (937-41-7); triethylene glycol diacrylate (1680-21-3); decyl acrylate (2156-96-9); stearyl acrylate (4813-57-4); pentaerythritol tetraacrylate (4986-89-4); diethylene glycol monoacrylate (13533-05-6); and 1,3-propanediol diacrylate (24493-53-6) (EPA 2004c).

Additional information was available on two of the above compounds. Triethylene glycol diacrylate, applied at 2.5 mg, twice weekly for 80 weeks, produced skin tumors (Williams, 2004). In a 78 week dermal carcinogenicity study, doses of 0.05, 0.1, and 0.5% triethylene glycol diacrylate were applied 5 days a week to 70 male C3H/HeNHsd mice per group, and dermatitis, acanthosis and hyperkeratosis, and intracorneal pusticules were the only toxic effects observed (Van Miller *et al*., 2003). Pentaerythritol tri/tetraacrylate (25% tri; 65% tetra), applied at 3 mg twice weekly for life did not produce skin tumors (Williams, 2004).

The chemicals identified in Table 3 below had information on carcinogenicity or mutagenicity in CCRIS. Three also had information posted with the European Chemicals Bureau; information on toxicity from this source is also included, as indicated.

The NTP has conducted or is presently conducting the following studies on trimethylolpropane triacrylate:

- 2 week, 13 week, and 2 year dermal studies in B6C3F1 mice and Fischer 344 rats
- 26 week dermal studies in TGAC (FVB/N) hemizygous mice
- Developmental studies in which the material was administered by gavage to rats and rabbits
- Immunology studies via the dermal route in B6C3F1 and BALB/C mice
- Micronucleus assay (negative)
- Salmonella (on test)
- Chemical disposition studies (intravenous and topical application) in mice and rats
- *In vitro* testing (bovine corneal opacity and permeability assay; cytotoxicity using the Epiderm™ skin model and MTT reduction in primary rat hepatocytesl and the neutral red uptake bioassay.

Although information has been presented to the Board of Scientific Counselors according to the NTP website, final technical reports on the cancer studies have not been prepared as of September 2005. A draft report posted on the NTP website does describe additional studies of interest that were not found in the IUCLID dataset or CCRIS. In a study conducted by Celanese Corporation in 1985, 5% trimethylolpropane triacrylate was administered to the shaven backs of 50 mice for 80 weeks. After 80 weeks, all surviving mice were sacrificed and 10% were examined histopathologically. Although acanthosis and epilated skin were observed, no skin tumors were reported. The draft report also cites another Celanese study that described trimethylolpropane triacrylate as not mutagenic in *Saccharomyces cerevisae*.

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Table 3. Information on Acrylates Removed from EPA's Master List

Chemical	CAS No.	Mutagenicity Data	Carcinogenicity Data
Acrylic acid	79-10-7	Ames battery: negative Mouse lymphoma assay: positive Chinese hamster ovary (CHO) cells (HPRT locus): negative CHO cells with and without S-9: chromosomal aberrations (CA) observed L5178Y mouse lymphoma cells and Chinese hamster lung (CHL) cells (S-9 not added): CA observed Rat hepatocytes and Syrian hamster embryo (SHE) cells (S-9 not added): unscheduled DNA synthesis (UDS) not observed SHE cells (S-9 not added): negative for micronuclei and cell transformation *In vivo* CA assays in rats: negative *Drosophila melanogaster*: negative for sex-linked recessive lethal mutations Dominant lethal assay: negative	C3H/HeJ mice, applied to skin 3 times a week for life: negative C3H/HeN HsdBR and Hsd:(ICR)GR mice, applied to skin 3 times a week for 21 months: negative Wistar rats, 26-28 month drinking water study, 120, 400, or 1,200 ppm: negative
Methyl methacrylate	80-62-6	Ames battery: negative Gene mutation assay with *Salmonella typhimurium* TM677: weak effect with S-9, negative without S-9 CHO cells: CA were observed at cytotoxic doses and a marginally positive response for sister-chromatid exchanges (SCE) was observed L5178Y mouse lymphoma cells (S-9 not added): CA were oberved	F344 rats and B6C3F1 mice, inhalation, 250 to 1,000 ppm for 102 weeks: negative for cancer but toxicological effects to the lung. Golden hamsters, inhalation 102-1,640 mg/m^3 for 78 weeks: no effects in nasal cavity F344 rats, inhalation, 25-400 ppm for 2 years: degenerative, hyperplastic, metaplastic, and inflammatory lesions of

		Mouse lymphoma assay: positive *In vivo* micronucleus assay: negative *In vivo* assay for CA: produced inconclusive findings Dominant lethal assay: negative	olfactory or respiratory epithelia at 2 highest doses Dogs and rats: oral, 2 years: negative in studies not conforming with current guidelines Retrospective mortality study of workers in acrylic sheet manufacture: increased risk of colon cancer Retrospective mortality study of acrylic fiber production plant workers; small increase in respiratory cancer
Neopentyl glycol diacrylate	2223-82-7	5 strain Ames battery with and without S-9: negative	
Diethylene glycol diacrylate	4074-88-8	5 strain Ames battery with and without S-9: negative	
Trimethylolpro-pane triacrylate	15625-89-5	4 strain Ames battery: positive in TA1535 with S-9; negative in other tests Mouse lymphoma L5178Y (TK+/TK-) assay: positive without S-9 but negative with S-9 CHO cells (HGPRT locus): positive with and without activation	
Tetraethylene glycol diacrylate	17831-71-9	4 strain Ames battery with and without S-9: negative Mouse lymphoma L5178Y (TK+/TK-) assay without S-9: positive CHO cells (HRPT locus) without S-9: positive	
Isooctyl acrylate	29590-42-9	5 strain Ames battery: negative with and without S-9 Mouse lymphoma L5178Y (TK+/TK-)	Male C3H mice, monomer applied to skin 3 times a week for lifetime: negative

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		assay: negative with and without S-9 *Saccharomyces cervisia*e: negative with and without S-9 Mouse embryo cells without S-9, cell transformation not observed	

Source: CCRIS (2004); European Commission (2000a,b; 2002a,b)

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