United States
Environmental Protection
Agency

Pollution Prevention
and Toxics
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OPPT Chemical Fact Sheets

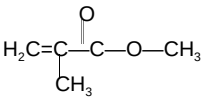
(METHYL METHACRYLATE) Fact Sheet: Support Document (CAS No. 80-62-6)

This summary is based on information retrieved from a systematic search limited to secondary sources (see Appendix A). These sources include online databases, unpublished EPA information, government publications, review documents, and standard reference materials. No attempt has been made to verify information in these databases and secondary sources.

I. CHEMICAL IDENTITY AND PHYSICAL/CHEMICAL PROPERTIES

The chemical identity and physical and chemical properties of methyl methacrylate are summarized in Table 1.

TABLE 1. CHEMICAL IDENTITY AND CHEMICAL/PHYSICAL PROPERTIES OF METHYL METHACRYLATE

Characteristic/Property	Data	Reference
CAS No.	80-62-6	
Common Synonyms	MME;2- methyl-2-propenoic acid, methyl ester; methyl-2-methyl-propenoate; Diakon	U.S. EPA 1985
Molecular Formula	$C_5H_8O_2$	
Chemical Structure		
Physical State	liquid	Keith and Walters 1985
Molecular Weight	100.1	
Melting Point	— 48 °C	U.S. EPA 1985
Boiling Point	100-101 °C @ 760 mm Hg	U.S. EPA 1985
Water Solubility	15 g/L @ 25 °C	U.S. EPA 1985
Density	0.939 g/mL$^{25/5}$	U.S. EPA 1985
Vapor Density (air = 1)	3.45	Keith and Walters 1985
Log K_{OC}	1.80 (calculated)	U.S. EPA 1985
Log K_{OW}	1.38 (measured)	Hansch and Leo 1985
Vapor Pressure	29.3 mm Hg @ 20 °C	U.S. EPA 1985
Reactivity	flammable; undergoes polymerization readily by light and heat	U.S. EPA 1985; Keith and Walters 1985
Flash Point	10 °C	Keith and Walters 1985
Henry's Law Constant	3.37 x 10^{-4} atm·m^3/mol	CHEMFATE 1994
Fish Bioconcentration Factor	6.6 (log 0.82) (calculated)	CHEMFATE 1994
Odor Threshold	0.2 mg/m^3 (detection)	Verschueren 1983
Conversion Factors	1 ppm = 4.09 mg/m^3 1 mg/m^3 = 0.244 ppm	U.S. EPA 1985

II. PRODUCTION, USES, AND TRENDS

A. Production

According to USITC (1994), CYRO Industries, DuPont Company*, and Rohm and Haas Company were the principal producers of methyl methacrylate in the United States in 1992. In 1992, the U.S. production volume of methyl methacrylate was estimated to be 1,205 million pounds (547,824 thousand kilograms) (Mannsville 1993; USITC 1994). The United States imported 10 million pounds and exported 120 million pounds of methyl methacrylate in 1992 (Mannsville 1993).

In 1993, Mannsville (1993) estimated CYRO Industries', ICI's, and Rohm and Haas' combined total capacity of methyl methacrylate as 1,435 million pounds (see Table 2). Table 2 shows the U.S. producers, plant locations, and plant capacities of methyl methacrylate in 1993. CYRO Industries has announced plans to expand its Fortier, Louisiana plant by 25 percent by the second quarter of 1995 (Mannsville 1993). Table 3 shows the domestic production capacity and production of methyl methacrylate.

TABLE 2. PRODUCERS OF METHYL METHACRYLATE AND THEIR CAPACITIES

Producer	Location	1993 Capacity (Millions of Pounds)
CYRO Industries	Fortier, LA	200
ICI Americas Inc*	Memphis, TN	320
ICI Americas Inc*	Beaumont, TX	125
Rohm and Haas	Deer Park, TX	790
TOTAL		1435

Source: Mannsville 1993.

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* In 1993, ICI acquired DuPont's Beaumont, Texas and Memphis, Tennessee plants, which manufacture methyl methacrylate.

TABLE 3. ESTIMATED U.S. PRODUCTION AND CAPACITY OF METHYL METHACRYLATE (Millions of Pounds)

Year	1990	1991	1992	1993(Projected)	1995(Projected)
Capacity	1310	1310	1435	1435	1485
Production	1182	1102	1084*	1088*	N/A

* Preliminary data.

N/A: Not available
Source: Mannsville 1993.

B. Uses

The principal application of methyl methacrylate is the production of acrylic plastics and resins for sheeting and molding compounds, which are used in construction, automotive/ transportation, consumer products, and industrial applications, and in making signs. Methyl methacrylate-butadiene-styrene (MBS) resins are used as impact modifiers for PVC used in making bottles. MBS resins help increase the capacity of a material to withstand blows or shocks.

Methyl methacrylate polymers and copolymers are used in waterborne, solvent, and solventless coatings. The largest surface coating application is exterior latex housepaint that is based on emulsions containing methyl methactylate. Table 4 provides the estimated 1993 domestic end use pattern for methyl methacrylate. Among the miscellaneous uses of smaller amounts of methyl methacrylate are those in dental restorations, in adhesive cements, and surgical (e.g., bone) implants.

TABLE 4. END USE PATTERN OF METHYL METHACRYLATE--1993 ESTIMATE

Derivative (Typical Standard Industrial Classification (SIC) Code)[1]	Percent
Cast and Extruded Sheet (SIC 2821)	30
Molding and Powder Resins (production, SIC 2821)	20
Protective Coatings (SIC 2851)	20
Impact Modifiers (SIC 2821)	12
Emulsion Polymers (SIC 2851)	9
Miscellaneous (no applicable SIC(s))	9

Source: Mannsville 1993.

[1] The Standard Industrial Classification (SIC) code is the statistical classification standard for all Federal economic statistics. The code provides a convenient way to reference economic data on industries of interest to the researcher. SIC codes presented here are not intended to be an exhaustive listing; rather, the codes listed should provide an indication of where a chemical may be likely to be found in commerce.

C. Trends

The United States consumption of methyl methacrylate grew at an average annual rate of five percent from 1983 to 1990. Demand for methyl methacrylate declined, however, in the 1990s due to the faltering of the automobile and construction industries. It is anticipated that demand for traditional rigid methacrylates will grow by one to two percent more than the GNP in the future. Overall, future demand for the methyl methacrylate monomer should grow four to five percent in 1994 with longer term growth at three to four percent per year. Demand for acrylic emulsions in aqueous systems are growing, replacing systems that contain volatile solvents in paints and adhesives. The markets for methyl methacrylate-butadiene-styrene (MBS) impact modifiers for PVC and twelve-inch video disks to record movies and music are expected to grow. Solvent-based applications of methylmethacryalte are declining.

III. ENVIRONMENTAL FATE

A. Environmental Release

Methyl methacrylate is released to ambient air from production facilities, end-product manufacturers and storage (U.S. EPA 1985). No atmospheric monitoring information was found for methyl methacrylate in the secondary sources searched; however, the chemical has been detected in the workplace under various conditions. In a polystyrene production plant, time-weighted-average (TWA) concentrations of 66 and 169 parts per billion (ppb) methyl methacrylate were detected in the workers' breathing zones and the air of the workplace, respectively; maximum breathing zone and workplace concentrations were 378 and 3300 ppb, respectively. At five US plants manufacturing polymethyl methacrylate, 8-hour TWA levels of methyl methacrylate were measured at 16 to 360 mg/m^3 (3.9-87.84 ppm) (U.S. EPA 1985).

Of 204 heavily industrialized sites tested in the Chicago and Illinois River basin area, only one surface water sample contained methyl methacrylate (concentration of 10 ppb) (U.S. EPA 1985). Methyl methacrylate (10 ppb) has been detected in one of nine sites monitored in Lake Michigan, in a few US drinking waters (<1 ppb), and in commercial deionized charcoal-filtered water (U.S. EPA 1985).

In 1992, environmental releases of the chemical, as reported to the Toxic Chemical release inventory by certain types of US industries, totaled about 2.8 million pounds, including 2.6 million pounds to the atmosphere, 220 thousand pounds to underground injection sites, 35 thousand pounds to surface water, and 4 thousand pounds to land (TRI92 1994).

B. Transport

Limited information was found regarding the atmospheric transport of methyl methacrylate. Given the water solubility of methyl methacrylate, some removal of methyl methacrylate from the atmosphere is expected to occur through dissolution into rain droplets (U.S. EPA 1985). Adsorption onto aerosols is not a likely transport process (U.S. EPA 1985).
Based on the Henry's law constant (3.37×10^{-4} $atm \cdot m^3/mol$ [CHEMFATE 1994]) and the vapor pressure of methyl methacrylate, volatilization from aqueous media is expected to be a significant transport mechanism for the chemical (U.S. EPA 1985). Log K_{OC} (1.80) and log K_{OW} (0.79) values suggest that sorption of methyl methacrylate onto particulate matter in aquatic media is not a significant removal mechanism.

No information was found on the transport of methyl methacrylate in soil. Based on the expected behavior of the chemical in water and air, it is likely to undergo significant evaporation from soil and, in cases where the evaporation or biodegradation of the chemical is delayed (such as near a spill or a dumpsite where the concentration may be higher), methyl methacrylate may leach into groundwater (U.S. EPA 1985).

C. Transformation/Persistence

1. Air — The major removal processes for methyl methacrylate in air are reaction with NO_X and direct photolysis (U.S. EPA 1985). In a smog chamber, the reaction of methyl methacrylate with NO_X proceeded with half-lives of 2.7 days and >3 days when the methyl methacrylate-to-NO_X ratios were 1:2 and 1:20, respectively (U.S. EPA 1985). At the methacrylate-to-NO_X ratio of 1:2, a maximum amount of ozone of 0.73 ppm was formed in 4.4 hours, whereas at the ratio of 1:20, 0.2 ppm were formed in 1.4 hours.

   At wavelengths greater than 300 nm, the photolysis of 2 mL methyl methacrylate in 4 mL dioxane produced 0.43% of the polymer in 1.2 hours. No information was found for the photolysis of methyl methacrylate in direct or simulated sunlight, but it is likely that ketonic compounds may accelerate the photoreaction (U.S. EPA 1985).

2. Water — Experiments conducted under biological treatment conditions suggest that the biodegradation of methyl methacrylate may be significant in the ambient aquatic environment (U.S. EPA 1985). The chemical: (a) was biodegradable (no quantitative estimates reported) with acclimatized sewage sludge; (b) exhibited a 47% theoretical oxidation to CO_2 in 10 days, using 19-day acclimatized sewage as microbial inoculum; and (c) underwent 100% degradation in ~20 hours with activated sludge as the source of microorganisms (U.S. EPA 1985).

   Methyl methacrylate may undergo photochemical reaction in aquatic media, particularly in surface waters (see section III.C.1); however, information on the photolysis of methyl methacrylate under environmental conditions was not found in the secondary sources searched.

3. Soil — No information was found on the fate of methyl methacrylate in soil. Based on the information on the degradation of methyl methacrylate in water, the chemical should undergo significant biodegradation in soil (U.S. EPA 1985).

4. Biota — The calculated log BCF of 0.82 and the low K_{OW} for methyl methacrylate suggest that the chemical will not bioconcentrate in aquatic organisms (U.S. EPA 1985).

IV. HEALTH EFFECTS

A. Pharmacokinetics

Methyl methacrylate is absorbed by the oral route, is metabolized first to methacrylic acid and ultimately to CO_2 via the Krebs cycle; metabolites are excreted mainly in expired air. Metabolites of methyl methacrylate may also be excreted in small amounts via the urine. Information indicates that methyl methacrylate does not acumulate in tissues.

1. Absorption — Animal studies demonstrate the rapid and extensive absorption of methyl methacrylate from the gastrointestinal (GI) tract following gavage (U.S. EPA 1985). ^{14}C-Methyl methacrylate, administered to rats at doses of 5.7 mg/kg, was expired as $^{14}CO_2$ at the rate of ~65% in 2 hours. Recovery of radioactivity for 10 days after oral dosing was 4.7% in urine, 2.7% in feces, 88% as $^{14}CO_2$, 0.1% unchanged ^{14}C-methyl methacrylate in expired air, and 6.6% in the carcass. Total recovery was 99.6%. No information was found on the absorption of methyl methacrylate via the respiratory tract, but systemic toxicity resulting from inhalation of the chemical indicates that it is absorbed (see sections IV.B and IV.C) by this route.

2. Distribution — Intravenously injected methyl methacrylate is cleared from the blood within minutes (U.S. EPA 1985). This, along with its metabolism (see section IV.A.3), suggests a low potential for the localization of methyl methacrylate in the tissues. Ten days after oral or intravenous dosing of rats with radiolabeled methyl methacrylate, small amounts of radioactivity, presumably from a radiolabelled metabolite, were detected only in liver and fat tissues (U.S. EPA 1985).

3. Metabolism — The results of animal studies with radiolabeled methyl methacrylate suggest that the chemical undergoes hydrolysis to methacrylic acid, which is converted into its coenzyme A ester (U.S. EPA 1985). The coenzyme A ester then enters the tricarboxylic acid cycle and is oxidized to carbon dioxide in the Krebs cycle. Other metabolites of methyl methacrylate that have been identified in the urine include methyl malonate, succinate, hydroxyisobutyrate, and 2-formyl propionate, with small amounts of the mercapturic acid, thioether, and the parent compound (ACGIH 1991). In addition, glutathione conjugation may play a minor role in the metabolism of methyl methacrylate (U.S. EPA 1985).

4. Excretion — Rats treated orally with labeled methyl methacrylate excreted ~65-88% of the administered doses as $^{14}CO_2$ in expired air (U.S. EPA 1985). Methyl methacrylate and its metabolites have also been detected in the urine (see section IV.A.3).

B. Acute Effects

Methyl methacrylate is of low acute toxicity to humans. Workers exposed to high concentrations of the chemical have experienced irritation of the skin, the eyes, and mucous membranes. Methyl methacrylate is also of low acute toxicity to animals exposed orally, by inhalation, or by percutaneous application.

1. Humans — Methyl methacrylate has been associated with skin, eye, and mucous membrane irritation (ACGIH 1991). In an occupational study, irritation (unspecified as to type) occurred when concentrations of methyl methacrylate reached 170 to 250 ppm (695 to 1022 mg/m^3); generally, workers tolerated levels of about 200 ppm without complaint, whereas 2300 ppm was intolerable (ACGIH 1991). (Note: to give the reader a rough approximation of approximate dose, the concentration of 170 ppm converts roughly to an intake of 99 mg/kg over an 8-hour workday[2]). In contrast, another study reported irritation of the mucous membranes at 125 to 200 ppm and suggested a tolerance level of 12 ppm (ACGIH 1991).

2. Animals — Oral LD_{50} values for methyl methacrylate are as follows: 7.8 to 9.4 g/kg for rats, 4.7 g/kg for dogs, 5.2 g/kg for mice, 5.9 g/kg for guinea pigs, and 6.6 g/kg for rabbits (U.S. EPA 1985).

   Inhalation LC_{50} values for methyl methacrylate are as follows: 19,000 mg/m^3 (4636 ppm) (8 hours) for rats, rabbits, and guinea pigs, 29,045 mg/m^3 (7087 ppm) (4 hours) for rats, and 13,100 mg/m^3 (3196 ppm) (2 hours) to 164,220 mg/m^3 (40,070 ppm) (27 minutes) for mice (U.S. EPA 1985). Non-lethal effects observed in rats exposed by inhalation to methyl methacrylate include GI irritation, dyspnea, upper respiratory tract irritation, and decreased GI motor activity at levels between 2000 ppm and 4000 ppm (8 to 18 g/m^3) (U.S.EPA 1990).

   The percutaneous LD_{50} is 7.5 g/kg for rabbits (U.S. EPA 1985). Methyl methacrylate is a potent skin sensitizer in guinea pigs, causing local necrosis and inflammation (ACGIH 1991).

C. Subchronic/Chronic Effects

Workers exposed to methyl methacrylate experienced allergic responses. Methyl methacrylate is a potent skin sensitizer in laboratory animals. In animals, the main effects of oral and inhalation exposures to moderate to high doses/concentrations of methyl methacrylate for 3 months to 2 years include local effects such as stomach ulcers and damage to the olfactory epithelium and lungs. Repeat inhalation exposure to large amounts of methyl methacrylate also adversely affect the liver, the spleen and bone marrow.

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[2] For dose comparison purposes this has been calculated by multiplying 695 mg/m^3 by 0.14 (the calculated occupational 8-hour workday breathing rate, 10 m^3, divided by the assumed adult body weight, 70 kg and assuming 100% absorption) to obtain the dose in mg/kg.

1. Humans — Allergic responses have been reported following direct contact with methyl methacrylate (U.S. EPA 1985). Exposure to methyl methacrylate induced hypersensitivity in 10% of dental technicians studied (total number not available) after 2-14 years of contact (U.S. EPA 1985).

   The following effects of methyl methacrylate have been noted in humans exposed under various conditions (exposure levels and durations not available): alterations of arterial pressure or heart rate in patients with total hip joint replacement requiring intraosseous application of methyl methacrylate; allergic sensitization; allergic contact eczema in dentists, dental technicians, and orthopedic surgeons; sore mouth in patients with dentures; headache and irritation of the eyes and respiratory tract resulting from exposure to vapors during surgery; effects on circulation, blood pressure, and bone metabolism; cardiovascular changes due to direct toxic effect of methyl methacrylate on the myocardium; and effects on renal function (U.S. EPA 1985).

2. Animals — Groups of 25 male and 25 female Wistar rats received drinking water containing 0, 6, 60, or 2000 ppm methyl methacrylate for five months (U.S. EPA 1985). At the start of the 5th month, the 6 and 60 ppm levels were increased to 7 and 70 ppm and treatment with all doses was continued for 2 years. Males and females in the high-dose group exhibited a transient weight depression at weeks 1 to 3 and high-dose females had an increased kidney-to-body weight ratio compared with controls. There were no differences in mortality among the groups and there were no treatment-related microscopic abnormalities.

   White rats (50/group) given average oral (gavage) doses of 114.6 mg/kg two days/week methyl methacrylate for 3, 5, or 8 months developed ulcers of the glandular epithelium of the stomach and irreversible liver lesions that became more severe as the duration of exposure increased (U.S. EPA 1985). Reversible lesions in the glomeruli were also noted.

   Beagle dogs given capsules containing 1437 ppm (TWA; equivalent dietary level) methyl methacrylate for 2 years exhibited slightly decreased weight gain, but those given $\leq$100 ppm showed no significant effect (U.S. EPA 1985).

   F344/N rats and B6C3F$_1$ mice (10 males and 10 females/group) inhaled 500, 1000, 2000, 3000, or 5000 ppm methyl methacrylate 6 hours/day, 5 days/week for 14 weeks (NTP 1986). No deaths occurred at the two lowest doses, but body weights were reduced. Mortality was dose-related at the highest doses (100% of the rats and 80% of the mice died at 5000 ppm). Both rats and mice had a dose-dependent increase in necrosis and sloughing of the olfactory epithelium. In addition, male rats exposed to 5000 ppm had follicular atrophy of the spleen and bone marrow atrophy. The mice displayed metaplasia (all exposed) and inflammation in the nasal turbinates, renal effects (males only) that included cortical necrosis and tubular degeneration with focal mineralization, and hepatic necrosis.

Exposure of white rats (50/group) to 12 ppm (49 mg/m^3) for 3 months (20 exposures), 5 months (41 exposures), or 8 months (63 exposures) produced histological changes in the lungs that were characterized by interstitial infiltrates, thickening of the septa, and marked alveolar desquamation (no other experimental details were available) (U.S. EPA 1985). Reversible lesions were observed in the liver and kidney.

Sprague-Dawley rats (25/group) were exposed to methyl methacrylate vapor concentrations of 0 and 116 ppm (475 mg/m^3) 8 hours/day, 5 days/week for 3 months or 6 months (U.S. EPA 1985). Compared with the controls, the treated animals had lower lung and spleen weights at 3 months and significantly lower body weights and fat content at 3 and 6 months. Rats exposed for 6 months also had significantly decreased "average small intestinal transit performance" (U.S. EPA 1985).

Groups of 56 male and 56 female Golden hamsters and groups of 70 male and 70 female Fischer rats were exposed by inhalation to 25, 100, or 400 ppm (102, 409, or 1638 mg/m^3) 6 hours/day, 5 days/week for 78 weeks (hamsters) or 104 weeks (rats). No treatment-related effects were noted in the hamsters; the rats had a slightly increased incidence of mild rhinitis, but no other effects (U.S. EPA 1985).

Groups of 50 male F344/N rats and 50 male and 50 female B6C3F$_1$ mice were exposed by inhalation to methyl methacrylate concentrations of 0, 500, or 1000 ppm, and groups of 50 female F344/N rats were exposed to concentrations of 0, 250, or 500 ppm for two years (NTP 1986). Exposure to methyl methacrylate was associated with concentration-related inflammation of the nasal cavity and degeneration of the olfactory sensory epithelium in exposed male and female rats and mice; epithelial hyperplasia of the nasal cavity was also observed in exposed mice.

Effects of inhaled methyl methacrylate noted in animal studies of shorter durations include: liver enzyme changes in mice exposed to 100 ppm for 6.6 days, lung damage in rats exposed to 1000 ppm for 56 hours, liver and kidney degeneration and death in dogs exposed to 1400 ppm for 1.5 hours/day for 8 days (U.S. EPA 1985).

D. Carcinogenicity

Epidemiology studies have suggested a causal relationship between exposure to methyl methacrylate and an increase in the incidence of cancer of the rectum and colon; however, the studies to date are inconclusive. There was no evidence for the carcinogenicity of methyl methacrylate administered to animals for up to two years, orally, by inhalation, and by skin painting. IARC has categorized methyl methacrylate as a Group 3 (not classifiable as to its carcinogenicity to humans) carcinogen.

1. Humans — Several epidemiological studies have suggested a causal relationship between exposure to mixtures of ethyl acrylate/methyl methacrylate and increased relative risk of cancer of the colon and rectum (U.S. EPA 1985); however, when analyses were broken down according to estimated accumulated "dose" and latency (the number of years between first exposure and death), a definite causal relationship could not be established.

   IARC (1987) has placed methyl methacrylate in Group 3 (not classifiable as to its carcinogenicity to humans), based on inadequate evidence of carcinogenicity in humans and animals.

2. Animals — Groups of 25 male and 25 female Wistar rats received drinking water containing 0, 6, 60, or 2000 ppm methyl methacrylate for five months (U.S. EPA 1985). At the start of the 5th month, the 6 and 60 ppm levels were increased to 7 and 70 ppm, and treatment was continued for 2 years. There were no differences in mortality among the groups and there were no treatment-related microscopic abnormalities.

   Groups of 50 male F344/N rats and 50 male and 50 female B6C3F$_1$ mice were exposed by inhalation to methyl methacrylate concentrations of 0, 500, or 1000 ppm, and groups of 50 female

F344/N rats were exposed by inhalation to concentrations of 0, 250, or 500 ppm for two years (NTP 1986). The NTP concluded that there was no evidence for the carcinogenicity of methyl methacrylate for male and female rats and male and female mice, under the conditions of the study.

Groups of 56 male and 56 female Golden hamsters and groups of 70 male and 70 female Fischer rats were exposed by inhalation to 0, 25, 100, or 400 ppm (102, 409, or 1638 mg/m^3) 6 hours/day, 5 days/week for 78 (hamsters) or 104 (rats) weeks. There was no increased incidence of neoplasm in treated animals compared with controls of either species (U.S. EPA 1985).

Skin painting with methyl methacrylate 3 times/week for 4 months did not produce skin tumors in 10 Wistar rats observed for the remainder of their lifespan (U.S. EPA 1985).

E. Genotoxicity

Genotoxicity data for methyl methacrylate are mixed. The chemical was positive, with S9, in *Salmonella typhimurium* (strain TM677) forward mutation assay and produced chromosomal aberrations in rats exposed to vapor concentrations of 1000 or 9000 ppm (4,094 or 36,846 mg/m^3) 6 hours/day for 5 days; there was no effect at 100 ppm. Methyl methacrylate was positive in hamster fibroblasts in culture medium containing 0.0065 mg/mL methyl methacrylate (U.S. EPA 1985). Methyl methacrylate was negative in *Salmonella typhimurium* strains TA1535, TA1537, TA1538, TA100, and/or TA98 in the vapor phase, plate incorporation, or liquid incubation assays, with and without metabolic activation; and was negative in the micronucleus and dominant lethal assays in mice (U.S. EPA 1985).

F. Developmental/Reproductive Toxicity

Limited information suggest that women and men exposed to methyl methacrylate have experienced decreased sexuality. Complications during pregnancy have been reported as well. Methyl methacrylate administered (in some cases, at maternally toxic levels) to pregnant rats has induced fetal toxicity. Methyl methacrylate has caused elevated estrogen secretion in rats.

1. Humans — Women and men (unspecified as to number) chronically exposed to methyl methacrylate have experienced decreased sexuality; complications during pregnancy were also reported (U.S. EPA 1985). Doses and durations of exposures were not reported.

2. Animals — Groups of 30 pregnant rats were exposed by inhalation to concentrations of methyl methacrylate ranging from 102 mg/m^3 to 4094 mg/m^3 (25 to 1000 ppm) 5 hours/day on days 6 through 15 of gestation (U.S. EPA 1985). No malformations were observed in any group, but there was an increased incidence of early resorptions in the animals exposed to 4094 mg/m^3. The highest no-adverse effect level reported for the study is 409 mg/m^3 (100 ppm). In another study, pregnant Sprague-Dawley rats were exposed to 110,000 mg/m^3 (26,840 ppm) of methyl methacrylate vapor for 17 or 54 minutes/day on days 6-15 of gestation. There were dose-related significant decreases in maternal weight gain and food consumption during and after exposure to methyl methacrylate. The group exposed for 54 minutes/day had significantly increased incidences ($p<0.05$) of early fetal death, decreased fetal weight, decreased crown-rump length, hematomas, and delayed sternebral ossification, compared with two control groups. The group exposed for 17 minutes/day also had an increased incidence of delayed sternebral ossification ($p<0.05$).

   Methyl methacrylate did not produce adverse effects in fetuses of 18 dams exposed by inhalation to 5445 mg/m^3 for 2 hours 2 times/day on days 6-15 of gestation (U.S. EPA 1985). Rats (unspecified as to sex) inhaling 54 mg/m^3 methyl methacrylate continuously for 1 to 4 months exhibited increased estrogen secretion, which apparently increased the follicle-stimulating activity of the pituitary (U.S. EPA 1985).

G. Neurotoxicity

Dental technicians and factory workers exposed to methyl methacrylate developed nerve degeneration in their hands and "nervous disorders", respectively. Repeated exposure to high concentrations of methyl methacrylate resulted in central nervous system effects in several animal species.

1. Humans — Five of twenty dental technicians who handled methyl methacrylate daily complained of neurological problems in their hands (U.S. EPA 1985). Measurement of distal sensory conduction velocities in the technicians revealed mild axonal degeneration in the areas of the hand with the closest and most frequent methyl methacrylate contact.

   Eighteen workers, employed for an average of 12 years in a factory manufacturing polymethyl methacrylate sheet, were divided into two groups, the first with exposure to <410 mg/m^3 and the second with exposure to >410 mg/m^3. Both groups exhibited nervous disorders (no details were available) (U.S. EPA 1985).

   Among the one hundred and fifty two workers exposed to 2 - 200 mg/m^3 (0.5-50 ppm) methyl methacrylate there were several reported adverse effects, including headaches, pain in extremities, sleep disturbance, loss of memory, and irritability. An unspecified 'most' of these workers had been employed for ≥ 10 years (U.S EPA 1985).

2. Animals — Rats exposed by inhalation to 400 ppm (1784 mg/m^3) of methyl methacrylate for 60 minutes exhibited depressed multiple-unit electrical activity in the lateral hypothalamus and ventral hippocampus. Exposures of guinea pigs and mice to 10,000 to 11,000 ppm for 0.5 to 3 hours/day for 15 days resulted in CNS depression and death apparently due to respiratory arrest (ACGIH 1991). Lethal concentrations of methyl methacrylate (3000 and 5000 ppm administered for 14 weeks) produced cerebellar congestion and hemorrhage into the cerebellar peduncles, malacia, and gliosis in F344/N rats (NTP 1986).

V. ENVIRONMENTAL EFFECTS

Methyl methacrylate is toxic to fish and *Daphnia* and inhibited cell multiplication in microorganisms only at high concentrations. Toxicity values for aquatic organisms are greater than 100 mg/L. Methyl methacrylate is acutely toxic to terrestrial animals only when present at very high concentrations.

A. Toxicity to Aquatic Organisms

The 24-, 48-, and 96-hour LC_{50} values in soft water are 421-455, 338-455, and 159-160 mg/L methyl methacrylate, respectively, for *Pimephales promelas* (fathead minnow); 368, 358, and 232 mg/L, respectively, for *Lepomis macrochirus* (bluegill); and 423, 423, and 277 mg/L, respectively, for *Carassius auratus* (goldfish) (U.S. EPA 1985). For *Leuciscus idus* (golden orfe), the 48-hour LC_0, LC_{50}, and LC_{100} for methyl methacrylate were 320, 350, and 380 mg/L; for *Daphnia magna* (water flea) in the immobilization assay, the 24-hour EC_0, EC_{50}, and EC_{100} values were 502, 720, and 1042 mg/L (U.S. EPA 1985). The toxicity threshold levels of methyl methacrylate inhibiting cell multiplication were 37 mg/L (8 days of exposure) for *Scenedesmus quadricauda* (green algae), 100 mg/L (16 hours) for *Pseudomonas putida* (bacteria), and 450 mg/L (72 hours) for *Entosiphon sulcatum* (protozoa) (U.S. EPA 1985).

B. Toxicity to Terrestrial Organisms

No information was found in the available literature for the toxicity of methyl methacrylate to terrestrial organisms. The oral LD_{50} values are greater than 5g/kg for rats and for mice (U.S. EPA 1985). Methyl methacrylate is acutely toxic to terrestrial animals only at very high concentrations.

C. Abiotic Effects

The ozone-forming potential of methyl methacrylate (see section III.C.1 and U.S. EPA 1985) indicates that the chemical may contribute to the formation of photochemical smog.

VI. EPA/OTHER FEDERAL/OTHER GROUP ACTIVITY

The Clean Air Act Amendments of 1990 list methyl methacrylate as a hazardous air pollutant. Occupational exposure to methyl methacrylate is regulated by the Occupational Safety and Health Administration. The permissible exposure limit (PEL) is 100 parts per million parts of air (ppm) as an 8-hour time-weighted average (TWA) (29 CFR 1910.1000). In addition to OSHA, other federal agencies and groups may develop recommendations to assist in controlling workplace exposure. These agencies and groups (listed in Tables 5 and 6) should be contacted regarding workplace exposures.

TABLE 5. EPA OFFICES AND CONTACT NUMBERS INFORMATION ON METHYL METHACRYLATE

EPA Office	Statute	Phone Number
Pollution Prevention & Toxics	PPA[a]	(202) 260-1023
	EPCRA (§313/TRI)[b]	(800) 535-0202
	TSCA (§ 8A, §8D)[c]	(800) 554-1404
Air	Clean Air Act (§111, §112B)[d]	(919) 541-0888
Solid Waste &	RCRA (U Waste)[e]	(800) 535-0202
Emergency Response	CERCLA (RQ, 1000 pounds)[f]	(800) 535-0202
Water	Clean Water Act (§311)[g]	(202) 260-7588

[a]**PPA**: Pollution Prevention Act
[b]**EPCRA**: Emergency Planning and Community Right to Know Act of 1986
[c]**TSCA**: Toxic Substances Control Act
[d]**CAA**: Listed as hazardous air pollutant under § 112 of Clean Air Act [42 U.S.C. 7401 et seq.; NAAQS = National Ambient Air Quality Standards (40 CFR 50.4, 50.6, 50.8, and 50.9-50.12).
[e]**RCRA**: The Resource Conservation and Recovery Act of 1976, (codified as amended at 42 U.S.C. §6901 *et seq*.
[f]**CERCLA**: Comprehensive Environmental Response, Compensation, and Liability Act of 1980, as amended. **RQ**: level of hazardous substance, which, if equaled or exceeded in a spill or release, necessitates the immediate reporting of that release to the National Response Center (40 CFR Part 302).
[g]**CWA**: Clean Water Act. Regulates waters of the United States, including surface waters, ground waters, and wetlands [40 CFR Part 131 (1994)] (U.S. EPA 1994b).

TABLE 6. OTHER FEDERAL OFFICES/CONTACT NUMBERS FOR INFORMATION ON METHYL METHACRYLATE

Other Agency/Department/Group	Phone Number
American Conference of Governmental Industrial Hygienists (TLV-TWA, 100 ppm; 410 mg/m^3)[a]	(513) 742-2020
Agency of Toxic Substances & Disease Registry (Group 4, priority list 2)	(404) 639-6000
Consumer Product Safety Commission	(301) 504-0994
Food & Drug Administration	(301) 443-3170
National Institute for Occupational Safety & Health (TWA, 100 ppm; 410 mg/m^3; IDLH, 4000 ppm)[b]	(800) 356-4674
Occupational Safety & Health Administration (TWA, 100 ppm; 410 mg/m^3)[c] (Check local phonebook under Department of Labor)	

[a]**TLV-TWA**: Time-weighted-average concentration for a normal 8-hour workday and a 40-hour workweek to which nearly all workers may be repeatedly exposed without adverse effects (ACGIH 1993-1994).

[b]**TWA**: Time-weighted-average concentrations for up to a 10-hour workday during a 40-hour workweek; **IDLH**: immediate danger to life and health (NIOSH 1990, 1992).

[c]**TWA**: Time-weighted-average that must not be exceeded during any 8-hour work shift of a 40-hour workweek. Standard promulgated pursuant to the Occupational Safety and Health Act, 29 CFR 1910 (OSHA 1993).

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