PROPYLENE

This substance was considered by a previous Working Group, in February 1978 (IARC, 1979). Since that time, new data have become available, and these have been incorporated into the monograph and taken into consideration in the present evaluation.

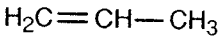
1. Exposure Data

1.1 Chemical and physical data

1.1.1 *Nomenclature*

Chem. Abstr. Serv. Reg. No.: 115-07-1
Replaced CAS Reg. No.: 676-63-1; 33004-01-2
Chem. Abstr. Name: 1-Propene
IUPAC Systematic Name: Propene
Synonyms: Methylethylene; 1-propylene

1.1.2 *Structural and molecular formulae and relative molecular mass*



C_3H_6 Relative molecular mass: 42.08

1.1.3 *Chemical and physical properties of the pure substance*

(*a*) *Description*: Colourless gas (American Conference of Governmental Industrial Hygienists, 1991; Lide, 1991)
(*b*) *Boiling-point*: –47.4 °C (Lide, 1991)
(*c*) *Melting-point*: –185.2 °C (Lide, 1991)
(*d*) *Density*: 1.9149 g/L at 25 °C, 760 mm Hg [101.3 kPa] (gas) (Eisele & Killpack, 1993); 0.5193 at 20 °C/4 °C (liquid under pressure) (Lide, 1991)
(*e*) *Spectroscopy data*: Infrared [prism, 6403] and mass spectral data have been reported (Weast & Astle, 1985; Sadtler Research Laboratories, 1991).
(*f*) *Solubility*: Soluble in acetic acid and ethanol (Lide, 1991)
(*g*) *Volatility*: Vapour pressure, 10 atm [1013 kPa] at 19.8 °C (Lide, 1991); relative vapour density (air = 1), 1.49 (Eisele & Killpack, 1993)
(*h*) *Stability*: Lower explosive limit (in air), 2.0 vol% (35 g/m^3) at 750 mm Hg [100 kPa] and 20 °C (Eisele & Killpack, 1993)

(*i*) *Octanol–water partition coefficient (P)*: log P, 1.77 (Hansch & Leo, 1979)
(*j*) *Conversion factor*: $mg/m^3 = 1.72 \times ppm^a$

1.1.4 *Technical products and impurities*

Propylene is available commercially in refinery, chemical and polymer grades (Anon., 1992). The grades of lower purity are generally used in close proximity to where they are produced, and the polymer grade dominates the commercial market. Polymer-grade propylene typically has a minimum purity of 99.5–99.8% and contains the following impurities: propane, methane, ethane, ethylene, propyne, butenes, propadiene, methylacetylene, butadiene, acetylene, diolefins, carbonyl sulfide, hydrogen, carbon monoxide, carbon dioxide, oxygen, nitrogen, water and sulfur (Dow Chemical Co., 1989; Phillips 66 Co., 1992; Eisele & Killpack, 1993; Exxon Chemical Co., undated a). Chemical-grade propylene typically has a minimum purity of 92.0–95.0% (Amoco Chemical Co., 1993; Eisele & Killpack, 1993; Exxon Chemical Co., undated b). Refinery-grade propylene generally contains 50–70% propylene admixed with other low relative molecular mass hydrocarbons (Eisele & Killpack, 1993).

1.1.5 *Analysis*

Atmospheric hydrocarbons, including propylene, can be detected and measured by capillary column gas chromatography and a flame ionization detector. The lower limit of measurement is 10 ppb by volume; this can be extended to 0.1 ppb by concentrating 100 ml of a gas sample in a freeze trap (Locke *et al.*, 1989). A variation on this method consists of preconcentration in a two-stage cryotrap system and an aluminium oxide-coated column; the limit of detection was 3 ppt (Schmidbauer & Oehme, 1985) or 2 pg (Matuska *et al.*, 1986). A similar method is based on sample enrichment on a solid sorbent, a zeolite, at room temperature, followed by heat desorption for gas chromatographic separation and flame ionization detection (Persson & Berg, 1989). Another method involves use of solid sorbent tubes in series (Tenax TA + Carbosphere S) and analysis by gas chromatography with electron capture detection in parallel with a tandem photoionization and flame ionization system; the limit of detection for propylene was 12 ppt (Reineke & Bächmann, 1985).

Methods have been developed for monitoring occupational exposures to propylene by determining a haemoglobin (Hb) adduct, *N*-(2-hydroxypropyl)valine (HOPrVal) of the metabolite, propylene oxide, using gas chromatography–mass spectrometry (Törnqvist *et al.*, 1986) and gas chromatography–electron capture detection (Kautiainen & Törnqvist, 1991).

1.2 Production and use

1.2.1 *Production*

Propylene is produced primarily as a by-product of petroleum refining and of ethylene production by steam cracking of hydrocarbon feedstocks (Schoenberg *et al.*, 1982). In

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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)

refinery production, propylene is formed as a by-product of catalytic cracking (and to a lesser extent thermal cracking) of gas oils. In steam cracking, a mixed stream of hydrocarbons ranging from ethane to gas oils is pyrolysed with steam. The product can be changed to optimize production of ethylene, propylene or other alkenes by altering feedstock, temperature and other parameters (Schoenberg *et al.*, 1982; Mannsville Chemical Products Corp., 1987; Eisele & Killpack, 1993).

Propylene has also been obtained by the catalytic dehydrogenation of propane in situations in which the propane value is low and derivatives are limited to local markets (Mannsville Chemical Products Corp., 1987).

Worldwide production of propylene in a number of countries is shown in Table 1. The percentage (by region) of worldwide production of propylene for use as a chemical intermediate is presented in Table 2. Production in 1990 by the countries of the European Union was 8005 thousand tonnes (European Commission, 1993).

Table 1. Worldwide production of propylene (thousand tonnes)

Country or region	Year					
	1982	1984	1986	1988	1990	1992
Canada	693	660	628	669	765	753
China	314	368	442	708	963	NR
France	1033	1203	1036	1496	1432	1795
Germany (western)	1478	1800	1403	1540	1827	2041
Italy	445	564	NR	NR	NR	NR
Japan	2565	2981	3167	3682	4215	4536
Mexico	158	208	232	281	NR	NR
Republic of Korea	NR	NR	307	351	608	1482
Taiwan	225	341	441	441	398	401
United Kingdom	769	974	862	1354	750	832
USA	5686	7058	7494	9627	9909	10 248[a]

From Scientific & Technical Information Research Institute of the Ministry of Chemical Industry of China (1984); Anon. (1985, 1987, 1989a, 1991, 1993); China National Chemical Information Centre (1993); Japan Petrochemical Industry Association (1993); NR, not reported

[a]Preliminary

Information available in 1991 indicated that propylene was produced by 24 companies in the USA, 16 in Japan, nine in Germany, four each in Brazil, Canada and France, three each in Belgium, China, the Netherlands and the United Kingdom, two each in the Republic of Korea, Spain and the former Yugoslavia, and one each in Argentina, Australia, Austria, Bulgaria, the former Czechoslovakia, Finland, India, Italy, Norway, Singapore, South Africa, Thailand and Venezuela (Chemical Information Services Ltd, 1991).

1.2.2 *Use*

Propylene is a major chemical intermediate. The most important derivatives of chemical and polymer grade propylene are polypropylene, acrylonitrile, propylene oxide, isopropanol

Table 2. Production of propylene for use as a chemical intermediate

Region	Production (%)		
	1980	1984	1990
North America	36.5	34.2	33.0
South America	3.5	4.3	4.8
Western Europe	31.3	31.9	30.2
Eastern Europe	9.1	10.1	8.9
Japan	14.2	13.2	13.9
Asia – Pacific	5.0	6.1	7.9
Others	0.4	0.2	1.3

From Eisele & Killpack (1993)

and cumene. Other commercial derivatives include acrylic acid and esters, oxo alcohols and aldehydes, epichlorohydrin, synthetic glycerine and ethylene–propylene copolymers. Use of polypropylene in plastics (injection moulding) and fibres (carpets) accounts for over one-third of US consumption; other applications are in wire insulating, film and blow moulding. Until the mid 1970s, polypropylene accounted for less than 25% of propylene consumption. Acrylonitrile, produced from chemical-grade propylene and ammonia and used as a precursor for fibres, plastics and nitrile rubber, makes up about 15% of US demand. Use patterns of propylene for several years in the USA are presented in Table 3; similar patterns are seen throughout the world (Schoenberg *et al.*, 1982; Anon., 1983, 1986; Mannsville Chemical Products Corp., 1987; Anon., 1989b, 1992; Eisele & Killpack, 1993). The pattern of use of propylene as a chemical intermediate in Japan in 1992 was: 48% for polypropylene, 15% for acrylonitrile, 9% for butanol and octanol (oxo alcohols), 7% for isopropanol, acetone and phenol, 6% for propylene oxide, 1% for ethylene–propylene rubber and 14% for other uses and exports (Japan Petrochemical Industry Association, 1993).

Refinery production accounts for about 20% of the chemical industry's consumption of propylene in Europe and for more than 40% in the USA. Refineries in the USA use about 75% of their propylene production in nonchemical applications, to prepare alkylates (octane enhancers) in gasoline production, to produce liquefied petroleum gas and as refinery heating gas (Eisele & Killpack, 1993).

1.3 Occurrence

Propylene in the environment arises from both anthropogenic and natural sources: as a natural product from vegetation, as a product of the combustion of organic matter (biomass burning, motor vehicle exhausts and tobacco smoke) and releases during the production and use of propylene (Altshuller, 1983; Tille *et al.*, 1985; Löfroth *et al.*, 1989; Rudolph *et al.*, 1989).

Table 3. Patterns of use of propylene as a chemical intermediate in the USA

Form of propylene	Use (%)			
	1983	1986	1989	1992
Polypropylene	27	36	38	39
Acrylonitrile	18	16	14	14
Propylene oxide	10	11	11	11
Cumene	8	8	10	10
Oxo alcohols	7	8	8	8
Isopropanol	7.5	6	6	7
Propylene oligomers	7	6	5	5
Acrylic acid	4	3	3	3
Miscellaneous[a]	12	6	5	3

From Anon. (1983, 1986, 1989b, 1992)
[a]Includes allyl chloride, ethylene/propylene elastomers, acrolein and exports

Annual US emissions of propylene from natural and anthropogenic sources have been calculated to range between 440 and 600 thousand tonnes (Altshuller, 1983; Middleton *et al.*, 1990). Annual emissions of propylene in western Europe between 35° and 50° N latitude have been calculated on the basis of data on source strength and emissions at 10 600 thousand tonnes and 2300 thousand tonnes, respectively (Tille *et al.*, 1985).

1.3.1 *Natural occurrence*

Propylene has been identified in emissions from vegetation. In a study of C_2–C_7 hydrocarbon emissions from 12 forest species near Baton Rouge, LA, USA, propylene was emitted from ash, elm, cypress and hackberry trees at levels ranging from 5 to 20 μg/kg foliage per h (Khalil & Rasmussen, 1992). The average emission rate of propylene from Chinese rice fields was estimated to be 0.4 μg/m^2 per h (Khalil *et al.*, 1990).

1.3.2 *Occupational exposure*

The National Occupational Exposure Survey conducted by the National Institute for Occupational Safety and Health in the USA between 1981 and 1983 indicated that 7300 US employees were potentially exposed to propylene at work (US National Institute for Occupational Safety and Health, 1993). Of this number, 88% were estimated to be exposed to propylene and 12% to materials containing propylene. The estimate is based on a survey of US companies and did not involve measurements of actual exposures.

Few data are available on levels of exposure to propylene in the workplace. In a study on exposures of firefighters, samples taken during the 'knockdown' phase of a fire contained a concentration of 8 ppm [13.8 mg/m^3] propylene; none was detected during the 'overhaul' phase (Jankovic *et al.*, 1991). In a laboratory study, propylene was identified as a thermal degradation product of polypropylene but was not detected in area samples taken in the

vicinity of injection moulding, extrusion and welding machines in four plants in which polypropylene was processed (Frostling *et al.*, 1984; Purohit & Orzel, 1988).

1.3.3 *Air*

Propylene concentrations measured in ambient air at rural and remote sites worldwide ranged from 0.02 to 8.3 μg/m^3 (Anlauf *et al.*, 1985; Colbeck & Harrison, 1985; Kanakidou *et al.*, 1989; Lightman *et al.*, 1990; Hov *et al.*, 1991; Mowrer & Lindskog, 1991; Satsumabayashi *et al.*, 1992).

In urban and polluted air, propylene concentrations typically range from 0.6 to 55 μg/m^3. Levels measured as monthly means in a number of cities around the world were: 0.4–18.7 ppb [0.7–32.3 μg/m^3] in Bombay, India (Netravalkar & Rao, 1984; Rao & Pandit, 1988); 0.3 ppb [0.5 μg/m^3] as an average of 192 samples in 1981in Tokyo, Japan (Uno *et al.*, 1985); 9.9 ppb as carbon (ppbC) [5.7 μg/m^3] in northwest England (Colbeck & Harrison, 1985); a range of 7–32 ppb by volume (ppbv) [12–55 μg/m^3] in Los Angeles, CA, USA (Grosjean & Fung, 1984) and 0.6–4.7 μg/m^3 in Chicago, IL, USA (Aronian *et al.*, 1989). Seinfeld (1989) reported a median propylene level of 7.7 ppbC [4.4 μg/m^3] for 39 US cities.

Although propylene is not a fuel component, it is present in motor vehicle exhaust as a result of fuel-rich combustion of hydrocarbon fuels (Stump *et al.*, 1989). Propylene accounted for 5% w/v of the non-methane hydrocarbon emissions from 67 Australian vehicles tested (Nelson & Quigley, 1984). In 1983, propylene emissions from petrol exhaust in the United Kingdom were estimated at 13 300 tonnes (Bailey *et al.*, 1990a,b). The following levels of propylene were determined in air samples representative of various traffic emissions in Sweden: 28 and 26 μg/m^3 (urban intersection), 6.5 and 3.8 μg/m^3 (fast suburban traffic) and 24 μg/m^3 (cold starts at a garage exit) (Löfgren & Petersson, 1992). Propylene concentrations of 13–160 μg/m^3 were determined in the Tingstad Tunnel in Göteborg, Sweden (Barrefors & Petersson, 1992).

Industrial emissions of propylene to the air in the USA in 1991 were reported to amount to 10 400 tonnes (US National Library of Medicine, 1993).

Cigarette smoke is also a significant source of exposure to propylene, as 1.3–1.4 mg propylene are released per cigarette (Persson *et al.*, 1988). Propylene concentrations of 40 and 70 μg/m^3 were found in two studies of tavern air during normal smoking conditions. The corresponding outdoor air concentration at the time was 6 μg/m^3 in both studies (Löfroth *et al.*, 1989).

A mean propylene concentration of 120 ppbC [69 μg/m^3] was determined in the indoor air of rural Nepali houses, where biomass combustion is the main source of energy (Davidson *et al.*, 1986).

The average atmospheric lifetime of propylene is estimated to be less than one day at low latitudes (Rudolph *et al.*, 1989). Propylene is subject to photochemical degradation by reaction with OH radicals (Tille *et al.*, 1985).

1.4 Regulations and guidelines

Propylene has been classified in several countries as an asphyxiant, because its presence at high concentrations in air lowers the available oxygen concentrations. Countries in which

it is classified as an asphyxiant include Australia, Belgium, Canada, Hungary, the Netherlands, the United Kingdom and the USA. Nevertheless, the major hazard is due to its inflammable and explosive character. No exposure limits have been established in most countries, but Switzerland established a time-weighted average occupational exposure limit of 17 500 mg/m^3 (about one-half the lower explosive limit) in 1987 (ILO, 1991; American Conference of Governmental Industrial Hygienists, 1993; UNEP, 1993).

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

2. Studies of Cancer in Humans

After the observation of an apparent cluster of colorectal cancers at a polypropylene manufacturing plant, Acquavella *et al.* (1988) carried out a cohort study of men who had been at the plant for six months or longer during 1960–85, allowing a 10-year induction period from first exposure. Seven incident colorectal cancers were ascertained (1.3 expected). Subsequently, a case–control study of adenomatous polyps and carcinoma *in situ* of the large bowel was carried out in the same workforce (Acquavella *et al.*, 1991). The occupational exposures of 24 cases identified in a screening programme were compared with those of 72 controls who had been found to be free of polyps. Propylene was handled at the plant, along with various other chemicals, but neither of the reports classified subjects according to whether they would have been exposed to propylene. They do not allow assessment of risk in relation to propylene exposure specifically.

3. Studies of Cancer in Experimental Animals

3.1 Inhalation

3.1.1 *Mouse*

Groups of 49–50 male and 50 female B6C3F1 mice, 9–10 weeks old, were exposed by inhalation to air containing 0 (control), 5000 or 10 000 ppm (8600 or 17 200 mg/m^3) propylene (purity, > 99%) for 6 h per day on five days per week for 103 weeks. The highest concentration of propylene was limited to 10 000 ppm because of the risk of explosion. Animals were killed at 104 weeks or when moribund. Survival of exposed and control mice was comparable. After week 59 of the study, the mean body weights of high-dose male mice were slightly lower (5%) than those of controls; the weights of animals in other exposed groups were comparable with those of the controls. Haemangiosarcomas were found in one low-dose male mouse (liver), two high-dose male mice (spleen) and three high-dose female mice (subcutis, spleen and uterus). Haemangiomas were found in one low-dose and one high-dose female mice (liver). In female mice, haemangiomas or haemangiosarcomas at all sites (combined) occurred in 0/50 controls, 1/49 low-dose and 4/50 high-dose mice (p = 0.024, incidental tumour test for trend). The overall historical incidence in female

controls was 90/2537 (3.5%; range, 0–10%). The occurrence of uterine endometrial stromal polyps in female mice showed a positive trend (control, 0/47; low-dose, 0/47; and high-dose, 3/48; $p = 0.044$, Cochran-Armitage test); the mean historical control rate was 22/2411 (0.9%; range, 0–6%). In male mice, the incidence of alveolar/bronchiolar adenomas or carcinomas (combined) showed a negative trend (control, 16/50; low-dose, 4/49; and high-dose, 7/50; $p < 0.05$, Cochran-Armitage trend test). The historical incidence of alveolar/bronchiolar adenoma or carcinoma (combined) in male mice was 397/2380 (16.7%; range, 2–34%) (Quest *et al.*, 1984; US National Toxicology Program, 1985).

Groups of 100 male and 100 female Swiss mice, seven weeks old, were exposed by inhalation to air containing 0 (control), 200, 1000 or 5000 ppm (344 mg/m^3, 1720 mg/m^3 or 8600 mg/m^3, respectively) propylene (purity: 97.0% propylene, 2.9% [propane], 6.5 ppm ethylene, 2.5 ppm ethane, 1 ppm methane) for 7 h per day on five days per week for 78 weeks. All animals were kept under observation until spontaneous death [times unspecified]. No difference in body weight was observed between treated groups and controls. A slightly increased mortality rate was observed among male mice exposed to 5000 ppm [exact numbers and toxic effects unspecified]. No difference was reported in the incidences of tumours among the different groups (Ciliberti *at al.*, 1988). [The Working Group noted the incomplete reporting.]

3.1.2 *Rat*

Groups of 50 male and 50 female Fischer 344/N rats, 9–10 weeks old, were exposed by inhalation to air containing 0 (control), 5000 or 10 000 ppm (8600 or 17 200 mg/m^3) propylene (purity, > 99%) for 6 h per day on five days per week for 103 weeks. The highest concentration of propylene was limited to 10 000 ppm because of the risk of explosion. Animals were killed at 102 weeks or when moribund. Survival of exposed and control rats was comparable. Throughout most of the study, the mean body weights of exposed male and female rats were slightly lower (0–5%) than those of the controls, but the decrements were not related to concentration. An increased incidence of squamous metaplasia of the nasal cavity was observed in treated female rats (control, 0/49; low-dose, 15/50; high-dose, 6/50) and in low-dose male rats (control, 2/50; low-dose, 19/50; high-dose, 7/50). The incidence of epithelial hyperplasia of the nasal cavity was increased in high-dose female rats (control, 0/49; low-dose, 4/50; high-dose, 9/50). Inflammation of the nasal cavity occurred at increased incidences in low- and high-dose male and in high-dose female rats. No treatment-related increase in tumour incidence was observed (Quest *et al.*, 1984; US National Toxicology Program, 1985).

Groups of 100 or 120 male and 100 or 120 female Sprague-Dawley rats, 17 weeks old, were exposed by inhalation to air containing 0 (control), 200, 1000 or 5000 ppm (0, 344, 1720 or 8600 mg/m^3) propylene (purity: 97.0% propylene, 2.9% [propane], 6.5 ppm ethylene, 2.5 ppm ethane, 1 ppm methane) for 7 h per day on five days per week for 104 weeks. All the animals were kept under observation until spontaneous death [times unspecified]. A slightly increased mortality rate was observed among mid- and high-dose male rats [not otherwise specified]. No difference was found in the incidences of tumours among the different groups (Ciliberti *at al.*, 1988). [The Working Group noted the incomplete reporting.]

3.2 Carcinogenicity of metabolites

See the monograph on propylene oxide

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 on the absorption, distribution or excretion of propylene in humans.

Hb adducts of the metabolic propylene intermediate propylene oxide (see below) have been used to monitor internal dose (see the monograph on propylene oxide, p. 193). In nonsmokers not exposed occupationally to propylene, the background level of the 2-hydroxypropyl adduct to the N-terminal valine (HOPrVal) of Hb was found to be about 2 pmol/g Hb. It was estimated that smoking 10 cigarettes per day would induce an increment of 2 pmol/g globin (Törnqvist & Ehrenberg, 1990). Occupational exposure to propylene at 1 ppm [1.72 mg/m^3] (40-h time-weighted average) was assumed to be associated with an increment of 5 pmol/g Hb (Kautiainen & Törnqvist, 1991).

4.1.2 *Experimental systems*

(*a*) *Propylene*

Propylene was oxidized to epoxypropane (propylene oxide), yielding about equal amounts of the two enantiomers (R, 43–50%, S, 50–57%), in incubates (37 °C, pH 7.4) containing human liver microsomes, an NADPH-regenerating system and trichloropropylene oxide to inhibit epoxide hydrolase activity (Wistuba *et al.*, 1989).

The pharmacokinetics of inhaled propylene have been investigated in male Sprague-Dawley rats (Golka *et al.*, 1989) and CBA mice (Svensson & Osterman-Golkar, 1984) in closed exposure chambers, in which the atmospheric concentration–time course was measured after injection of a single dose into the chamber atmosphere. The uptake of propylene into the body of rats has been found to be low. Clearance due to uptake, reflecting the rate of transfer of propylene from the atmosphere into the organism, was 19 ml/min for one rat weighing 250 g. This value represents only 16% of the alveolar ventilation (117 ml/min; Arms & Travis, 1988). Most propylene inhaled into the lungs is exhaled again and does not reach the blood to become systemically available. Maximal accumulation of propylene in the same rat, determined as the thermodynamic partition coefficient for whole body:air (K_{eq} = $Conc._{animal}/Conc._{air}$), was only 1.6. At concentrations below 50 ppm [86 mg/m^3], the concentration ratio at steady-state whole body:air was even smaller (0.7), owing to metabolic elimination. At these concentrations, first-order kinetics were found, and clearance due to metabolism in relation to the concentration in the atmosphere was calculated to be 11 ml/min for a 250-g rat. Thus, at steady state, about 58% of systemically

available propylene is eliminated metabolically and 42% is eliminated by exhalation as the unchanged substance. No corresponding data have been published for mice. In rats and mice, the rate of propylene metabolism showed saturation kinetics, with maximal metabolic rates (V_{max}) of 0.17 μmol/(min×250 g bw) [1.7 mg/(h×kg bw)] and 8 mg/(h×kg bw) and atmospheric concentrations at $V_{max}/2$ of 260 ppm [447 mg/m^3] and 800 ppm [1376 mg/m^3], respectively.

Dahl *et al.* (1988) investigated the rates of propylene uptake in male Fischer 344/N rats exposed by nose only to a constant atmospheric concentration of 100 ppm [172 mg/m^3]. Normalized values of the rates of metabolism determined after 60 min of exposure in two independent experiments were 1.3 and 1.9 nmol/(kg bw×min×ppm) [3.3 and 4.8 μg/(kg bw×h×ppm)]. Under steady-state conditions, the amount taken up equals the amount metabolized. Consequently the two values are equivalent to the rates of metabolism at steady state and are in agreement with a value for male Sprague-Dawley rats of 4.4 μg/(kg bw×h×ppm), which can be calculated on the basis of the clearance of metabolism reported by Golka *et al.* (1989).

(b) Metabolites

Propylene oxide (see monograph, p. 193) is produced during the first step of propylene metabolism. Epoxidation by a reconstituted cytochrome $P450_{LM2}$ system including cytochrome P450 reductase proceeded with a concomitant, stereospecific (*trans* to the alkyl group) hydrogen/deuterium exchange from the aqueous phase when *trans*-1-deuteriopropylene in H_2O or propylene in D_2O (pH 7.4, 4 °C) was used (Groves *et al.*, 1986).

Both enantiomers of propylene oxide were found after incubation (37 °C, pH 7.4) of propylene with an NADPH-regenerating system and microsomes prepared from livers of male Wistar rats (control and phenobarbital-pretreated animals) and male NMRI/HAN mice (control, phenobarbital- and benzo[*a*]pyrene-pretreated animals). Epoxide hydrolase activity was inhibited in those incubates which contained trichloropropylene oxide. In incubations with liver microsomes of rats, about 30% of R and about 70% of S enantiomer were formed, whereas with mouse microsomes the amounts of the two enantiomers were nearly equal: R, 42–55%; S, 45–58%, depending on the pretreatment (Wistuba *et al.*, 1989).

After male Fischer 344/N rats were exposed by nose only to atmospheric propylene concentrations of 6 and 600 ppm [10.3 and 1032 mg/m^3], propylene oxide was determined in blood by gas chromatography–mass spectrometry (Maples & Dahl, 1991). Abrupt increases in the concentrations of propylene oxide in blood were reported to occur only a few minutes after beginning of exposure to either 6 or 600 ppm propylene. The concentrations of propylene oxide decreased rapidly after 10 min of exposure, to values about 200 times lower, and remained relatively constant over a further 50 min of exposure. The cytochrome P450 content was measured in microsomes prepared from liver, nasal ethmoturbinates and maxilloturbinates of rats after 0, 20 and 360 min of exposure. After 20 min of exposure to 600 ppm propylene, hepatic and nasal cytochrome P450 activities were reduced to about 70 and 50%, respectively, but returned to the control value (nasal cavity) or to even higher values (liver) after 360 min of exposure. It was speculated that a propylene-specific cytochrome P450 isozyme might be rapidly deactivated during exposure to propylene, resulting in reduced formation of propylene oxide (Maples & Dahl, 1991). In another study, mice were

exposed over 6–7 h to propylene at concentrations of 230, 680, 22 100 and 30 000 ppm [396, 1170, 38 000 and 51 600 mg/m^3], and the amounts of the metabolically formed propylene oxide were calculated, taking into account the saturable metabolism of propylene. The Hb adduct levels were then determined and plotted against the calculated amounts of propylene oxide, resulting in a linear curve. In further experiments, Hb adduct levels were measured after intraperitoneal administration of propylene oxide at doses of 0.065, 0.1 and 0.19 mg/kg bw. The plot of Hb adduct level *versus* administered dose of propylene oxide resulted in a linear curve with a slope almost identical to that obtained in the former plot (Svensson *et al.*, 1991).

In male Sprague-Dawley rats, propylene metabolism becomes increasingly saturated at concentrations above 50 ppm [86 mg/m^3] in the atmosphere, whereas propylene oxide metabolism is not saturable, at least up to 3000 ppm [5160 mg/m^3]. Kinetic data obtained following exposure to propylene and propylene oxide were used to calculate that the theoretical maximal body burden of propylene oxide gas from propylene is 71 nl/ml body tissue [0.16 mg/kg bw]. These calculations do not take into account detoxification in the cell that metabolizes propylene to propylene oxide (Golka *et al.*, 1989).

(*c*) *Haemoglobin adducts*

Hb adducts of propylene oxide in male CBA mice exposed to ^{14}C-propylene have been found at cysteine, histidine and N-terminal valine (Svensson & Osterman-Golkar, 1984; Svensson *et al.*, 1991). Exposure to 20 000 ppm propylene [34 400 mg/m^3] for 4 h/day on eight consecutive days resulted in an alkylation rate of 2200 pmol N$^{\tau}$-(2-hydroxypropyl)-histidine/g Hb per h and an estimated rate of formation of propylene oxide of 11 mg/(h×kg bw) (Svensson & Osterman-Golkar, 1984). A linear relationship was reported between the concentration of HOPrVal (240–1620 pmol/g Hb) and the amount of propylene oxide (0.3–1.3 mmol/kg bw [17.4–75.4 mg/kg bw]) formed in mice after they had inhaled propylene (230–30 000 ppm×7 h [2770–361 200 mg×h/m^3]). This relationship had almost the same slope as that obtained after intraperitoneal injection of ^{14}C-propylene oxide in saline (0.065–0.19 mmol/kg bw [3.8–11 mg/kg bw]). It was concluded that propylene oxide is the major primary metabolic product of propylene (Svensson *et al.*, 1991). [The Working Group noted, furthermore, that there is effective biotransformation of propylene to propylene oxide throughout the exposure period.]

HOPrVal in Hb was determined in female Fischer 344 rats and male and female Syrian hamsters exposed for six months to gasoline and diesel exhausts (mean atmospheric concentrations of propylene, < 0.1–0.72 ppm [< 0.17–1.24 mg/m^3]). Background values for HOPrVal were 9 pmol/g Hb in rats and 6 pmol/g Hb in hamsters. In hamsters, the levels of HOPrVal increased almost linearly with exposure dose and were higher in females than in males. HOPrVal adduct increments at the highest dose were similar in female rats (44 pmol/g Hb) and hamsters (47 pmol/g Hb) (Törnqvist *et al.*, 1988).

After a 4-h exposure of phenobarbital-pretreated male Sprague-Dawley rats to propylene (40% in air [688 g/m^3]), abnormal porphyrins were found to have accumulated in the liver. It was concluded that the porphyrins resulted from alkylation of the prosthetic haem group of cytochrome P450 during the oxidation of propylene that led to inactivation of the enzyme. An N-(2-hydroxypropyl) adduct at the pyrrole ring D was identified by nuclear

magnetic resonance analysis of alkylated porphyrins. *In vitro*, an NADPH-dependent reduction of total cytochrome P450 by 32% was measured in propylene-exposed (5% in air [86 g/m^3], 30 min) liver microsomes isolated from phenobarbital-pretreated male Sprague-Dawley rats (Kunze *et al.*, 1983).

Exposure of male Sprague-Dawley CD rats to 50 000 ppm [86 g/m^3] propylene for 4 h after treatment with phenobarbital, β-naphthoflavone or a combination of the two resulted in a decrease of about 40% in the control values for hepatic cytochrome P450 content. Similar exposure of polychlorinated biphenyls (Aroclor 1254)-treated rats to propylene led to a 60% reduction in the hepatic cytochrome P450 content for at least 24 h after the start of exposure. Furthermore, in liver microsomes prepared from these animals, a 20% reduction in hepatic microsomal aniline hydroxylase activity was observed in comparison with controls. In experiments *in vitro*, liver microsomes obtained from pretreated but not propylene-exposed animals were incubated with propylene (20% in air [344 g/m^3], 20 min, 37 °C) in the presence of NADPH. This treatment decreased the cytochrome P450 content to 70–80% of control values. In non-pretreated animals, propylene had no effect on cytochrome P450 concentrations (Osimitz & Conolly, 1985).

4.2 Toxic effects

The toxicology of propylene has been reviewed (Gibson *et al.*, 1987).

4.2.1 *Humans*

No data were available to the Working Group.

4.2.2 *Experimental systems*

(*a*) *Acute toxicity*

Inhaled propylene was not toxic to male Sprague-Dawley CD rats exposed for 4 h to an atmospheric concentration of 50 000 ppm [86 g/m^3]; however, hepatotoxic effects were seen in propylene-exposed (50 000 ppm, 4 h) animals which had been pretreated with Aroclor 1254. Hepatotoxicity was manifested by an increase in the liver:body weight ratio, by the presence of macroscopic focal areas of haemorrhage and by elevated activities of serum sorbitol dehydrogenase and alanine leucine transaminase. SKF-525A, an inhibitor of cytochrome P450-dependent metabolism, abolished these effects when given before propylene exposure. Reduction of the glutathione content by fasting did not influence the hepatotoxicity of propylene in Aroclor-pretreated rats. It was concluded that Aroclor might have induced a specific cytochrome P450 isozyme, or the same isozymes as those induced by phenobarbital or β-naphthoflavone, but to a greater extent. In either case, sufficient propylene may have been metabolized to cause hepatotoxicity. Alternatively, Aroclor may predispose the liver to damage caused by an otherwise innocuous level of propylene metabolism (Osimitz & Conolly, 1985).

(*b*) *Subchronic toxicity*

Exposure of male and female Fischer 344/N rats and male and female B6C3F1 mice for 14 days or 14 weeks to atmospheric propylene concentrations ranging from 625 to

10 000 ppm [1075 to 17 200 mg/m^3] did not induce toxic effects (US National Toxicology Program, 1985).

(*c*) *Chronic toxicity*

Male and female Sprague-Dawley rats were exposed to propylene at concentrations of 200, 1000 or 5000 ppm [344, 1720 or 8600 mg/m^3] for 7 h/day on five days/week for 104 weeks and male and female Swiss mice for 7 h/day on five days/week for 78 weeks. The mortality rate of male rats increased slightly after exposure to 1000 and 5000 ppm [1720 and 8600 mg/m^3] and that of male mice after exposure to 5000 ppm [8600 mg/m^3] (Ciliberti *et al.*, 1988).

In another study, male and female Fischer 344/N rats and male and female B6C3F1 mice were exposed for 6 h/day on five days/week for 103 weeks to atmospheric propylene concentrations of 5000 and 10 000 ppm [8600 and 17 200 mg/m^3]. Squamous metaplasia of the nasal cavity developed in females at both exposure concentrations (not dose dependent) and in males at the low exposure concentration, and epithelial hyperplasia developed in females exposed to the high concentrations. In males, inflammatory changes were seen in the submucosa and the lumen of the nasal cavity. According to the authors, all of the nasal cavity lesions reflected changes due to local irritation. No change was observed in the nasal cavity of mice of either sex, but the incidences of chronic focal renal inflammation were increased in both exposure groups (Quest *et al.*, 1984; US National Toxicology Program, 1985).

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 4 and Appendices 1 and 2)

Propylene did not induce gene mutation in *Salmonella typhimurium* TA100 exposed for 7 h to 20% propylene in air in either the presence or absence of an exogenous metabolic activation system. Exposure of L5178Y mouse lymphoma cells to 20–50% propylene in air for 4 h did not induce mutations at the *tk* locus in the absence of metabolic activation but did produce inconclusive results in the presence of an exogenous metabolic activation system from rat liver.

Alkylation of DNA at the N7 position of guanine was investigated in male CBA mice exposed to atmospheric propylene or uniformly labelled ^{14}C-propylene. The adduct levels were related to the concentration of propylene oxide (0.88 mmol/kg bw), calculated from the rate of propylene metabolism. Immediately after exposure to 107 MBq uniformly labelled ^{14}C-propylene (18.1 MBq/mmol propylene) for 7 h in a closed exposure chamber, in which the atmospheric concentration–time course was measured after injection of a single dose

Table 4. Genetic and related effects of propylene

Test system	Results[a]		Dose[b] (LED/HID)	Reference
	Without exogenous metabolic system	With exogenous metabolic system		
SA0, *Salmonella typhimurium* TA100, reverse mutation	–	–	336.0000[c]	Victorin & Ståhlberg (1988)
G5T, Gene mutation, mouse L5178Y cells, *tk* locus	–	?	840.0000[c]	McGregor *et al.* (1991)
BVD, Binding (covalent) to mouse DNA *in vivo*	+		0.0000	Svensson *et al.* (1991)
Protein binding				
BVP, Binding (covalent) to mouse haemoglobin *in vivo*	+		8000.0000, inhal. 4 h/d × 8 d	Svensson & Osterman-Golkar (1984)
BVP, Binding (covalent) to mouse protein *in vivo*	+		480.0000 inhal. 7 h	Svensson *et al.* (1991)

[a] +, positive; –, negative; ?, inconclusive (variable response within several experiments within an adequate study)
[b] In-vitro tests, μg/ml; in-vivo tests, mg/kg bw
[c] Atmospheric concentration in exposure chamber (μg/ml)

into the chamber atmosphere, the mice were killed and 2-hydroxypropyl–DNA adducts were measured. The values were: liver, 3000 pmol/g DNA; kidney, 3000 pmol/g DNA; and spleen, 2000 pmol/g DNA (Svensson *et al.*, 1991). [The Working Group noted that the results were based on low counts of radioactivity.]

5. Summary of Data Reported and Evaluation

5.1 Exposure data

Propylene is a major chemical intermediate, produced by catalytic or thermal cracking of hydrocarbons or as a by-product of petroleum refining. It is used mainly in the preparation of alkylates for gasoline and in the production of polypropylene, acrylonitrile, propylene oxide and a number of other industrial chemicals. Propylene is introduced into the atmosphere from natural and man-made sources, including emissions from vegetation, burning of organic material and incomplete combustion of fossil fuels, and from its production and use. Few data are available on levels of occupational exposure.

5.2 Human carcinogenicity data

No relevant data were available to the Working Group.

5.3 Animal carcinogenicity data

Propylene was tested by inhalation in two studies in mice and in two studies in rats. A slight increase in the incidence of vascular tumours was observed in female mice in one study. In one study in rats, no treatment-related increase in tumour incidence was observed. In two studies in mice and rats exposed by inhalation, insufficient information was provided to allow an assessment of carcinogenicity.

5.4 Other relevant data

In rats exposed to 50 ppm propylene, about one-sixth of the inhaled material is absorbed, of which almost one-half is exhaled again, unchanged. The remainder is eliminated metabolically, through oxidation to propylene oxide, which is subsequently either conjugated with glutathione or, to a smaller extent, hydrated by epoxide hydrolase. Oxidation is a saturable reaction mediated by cytochrome P450 enzymes, whereas no saturation concentration has been identified for the hydration of propylene oxide. There is, therefore, a maximal attainable tissue concentration of propylene oxide in rats. Oxidation of propylene can occur in the rat nasal epithelium, where irritation, hyperplasia and metaplasia have been described after chronic exposure.

No data were available on the genetic and related effects of propylene in humans.

Alkylation products of the metabolite, propylene oxide, were found in haemoglobin and in DNA from mice exposed to propylene by inhalation. Although insufficient data are

available to evaluate the genetic and related effects of propylene, its major metabolite, propylene oxide, is genotoxic in a broad range of assays.

5.5 Evaluation[1]

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

There is *inadequate evidence* in experimental animals for the carcinogenicity of propylene.

Overall evaluation

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

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

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