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Summary Review of the Health Effects Associated with Propylene

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SUMMARY REVIEW OF HEALTH EFFECTS ASSOCIATED WITH PROPYLENE

Health Issue Assessment

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Preface

The Office of Health and Environmental Assessment has prepared this health issue assessment to serve as a source document for EPA use. The health assessment was developed for use by the Office of Air Quality Planning and Standards to support decision making regarding possible regulation of propylene as a hazardous air pollutant under the Clean Air Act, as amended in 1977.

In the development of the assessment document, the scientific literature has been inventoried, key studies have been evaluated, and summary/conclusions have been prepared so that the chemical's toxicity and related characteristics are qualitatively identified. Observed effect levels and other measures of dose-response relationships are discussed, where appropriate, so that the nature of the adverse health responses is placed in perspective with observed environmental levels.

The relevant literature for this document has been reviewed through March 1988.

Information regarding sources, emissions, atmospheric transformation, ambient air concentrations, and human exposure is summarized to provide a preliminary indication of the potential presence of this substance in the ambient air. While the available information is presented as accurately as possible, it is acknowledged to be limited and dependent in many instances on assumption rather than specific data. This information is not intended and, therefore, should not be used as an exposure assessment by which to estimate risk to public health.

If a review of the health information indicates that the Agency should consider regulatory action for this substance, a considerable effort will be undertaken to obtain appropriate, more detailed information regarding sources, emissions, and ambient air concentrations. Such data will provide additional information for drawing regulatory conclusions regarding the extent and significance of public exposure to this substance.

Table of Contents

	Page
Figures and Tables	vi
Authors, Contributors, and Reviewers	vii
1. Background Information	1
1.1 Chemical Characterization and Measurement	1
1.2 Environmental Release, Transformation and Exposure	2
1.3 Environmental Effects	11
2. Health Effects	13
2.1 Pharmacokinetics and Metabolism	13
2.2 Biochemical Effects	13
2.3 Acute Toxicity	14
2.4 Subchronic Toxicity	15
2.5 Chronic Toxicity	15
2.6 Mutagenicity	16
2.7 Carcinogenicity	18
2.8 Other Health Effects	20
3. Summary	21
4. References	23

Figures

Number		Page
1-1	Daytime propylene reaction sequences in the urban troposphere	6
1-2	Hydroxyl radical-initiated gas-phase oxidation of propylene	7

Tables

Number		Page
1-1	Propylene concentrations in selected ambient air samples	4
1-2	Reactants and products in the propylene-NO_x-air photooxidation system	8
2-1	Incidence of epithelial changes in the nasal cavities of rats exposed to propylene via inhalation	16
2-2	Incidence of tumors in mice exposed to propylene via inhalation	19

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The information presented in this document is based on literature searches of databases and updates, the most recent of which was conducted during March 1988.

1. BACKGROUND INFORMATION

This overview provides a brief summary of the data available on the health effects of exposure to propylene. Emphasis is placed on determining whether there is evidence to suggest that propylene exerts effects on human health under conditions and at concentrations commonly experienced by the general public. Both acute and chronic effects are addressed, including general toxicity, mutagenicity, and carcinogenicity. To place the health effects discussion in perspective, this report also summarizes air quality aspects of propylene in the United States, including sources, distribution, fate, and concentrations associated with certain point sources.

1.1 Chemical Characterization and Measurement

Propylene (CAS No. 115-07-1) has the chemical structure $CH_3CH=CH_2$. It is a colorless gas with a molecular weight of 42.08. It is only slightly soluble in water (44.6 ml/100 ml at 20°C and 1 atm or 82.1 mg/100 ml) and has a high vapor pressure (10 atm at 19.8°C) (TDB; Schoenberg et al., 1982).

Although infrared spectrophotometry can be used to detect propylene in gaseous mixtures (International Agency for Research on Cancer, 1979), gas chromatography (GC) is the analytical method most commonly used to measure concentrations of the compound in environmental media and combustion gases. For the qualitative and quantitative determination, gas chromatography with flame ionization detection (GC/FID) and mass spectroscopy (MS) have been used extensively, e.g., Nelson and Quigley (1982) and Crow et al. (1982) for ambient air concentrations; Hoff et al. (1982) for pyrolysis products; and Yoshida et al. (1978) for polymer combustion gases. Cryogenic collection and the use of solid adsorbents have been used as sample preconcentration steps preceding GC analyses. The GC method used by Westberg et al. (1974) for the separation of C_2-C_6 hydrocarbons in ambient air uses a special chemically bonded stationary phase and has a detection limit of 0.5 ppb. Single run C_2-C_{10} HC analysis was accomplished using the open trap cryofocusing/GC-reinjection technique (Matuška et al., 1986). An average retention time of 4.322 minutes was reported for propylene.

Rudolph et al. (1981) developed a GC/FID method for determination of low levels of C_2-C_5 hydrocarbons in grab samples (0.5-2 cm^3) of air. Hydrocarbons in the samples are first concentrated on a packed pre-column with porous silica and

Carbosieve B kept at -35°C and then separated on a column packed with untreated porous silica (Spherosil XOB 075). The detection limit for propylene using this method is 4 ppt. A similar GC/FID method for determining C_2-C_8 hydrocarbons has recently been described by Reineke and Bachmann (1985). This method employs a three-detector setup and reproducibly quantifies propylene at concentrations as low as 12 ppt.

A GC/FID method, with detection limits of 2×10^{-12} M for gas and 5×10^{-8} ml for gas dissolved in 1 liter of sea water at standard temperature and pressure, was used by Swinnerton and Linnenbom (1967) to measure propylene concentrations in sea water samples. Frisch et al. (1982) used a gas radio chromatographic liquid scintillation spectrophotometric method to detect propylene in the smoke of cigarettes made with a tobacco filler treated with a radiolabeled insecticide.

1.2 Environmental Release, Transformation, and Exposure

Domestic production capacity of propylene, as of January 1, 1984, was estimated to be about 22.3 billion pounds annually, involving 37 companies operating at 61 plant sites (SRI International, 1984). The compound is produced as a by-product of petroleum refining and as a coproduct of ethylene manufacture. Propylene is used in both chemical and refinery applications. Refinery-generated propylene is the main source of the compound for nonchemical uses, whereas the ethylene coproduct material is used mainly in chemical applications. Chemical applications accounted for about 58 percent of domestic consumption of the compound in 1979; refinery applications accounted for the remaining 42 percent. Propylene is marketed in three grades: refinery (50-70 percent pure), chemical (90-96 percent pure), and polymer (>99 percent pure) (Chemical Economics Handbook, 1980; Schoenberg et al., 1982).

The current demand of 14.6 billion pounds of propylene annually for chemical applications is expected to grow at an average rate of 5 percent per year through 1988, to a level of 17.7 billion pounds annually (Chemical Marketing Reporter, 1984). Propylene is used both in the chemical and the petroleum industries as a monomer in the production of polypropylene (27 percent), and as a raw material in the production of acrylonitrile (18 percent), propylene oxide (9.7 percent), and cumene (7.9 percent of the consumption). Production of isopropyl alcohol, other alcohols, and numerous miscellaneous chemicals (e.g., propylene oligomers, butyraldehydes, acrylic acid, allyl chloride, and acrolein) account for the remainder of propylene chemical consumption (Chemical Economics Handbook, 1980; Schoenberg et al., 1982; Chemical Marketing Reporter, 1984).

Data on the current demand for propylene in refinery applications were not available. In refinery applications, propylene is used as a feedstock for high-octane blending components of gasoline (e.g.,

alkylate and polymer gasoline) and as a fuel (Chemical Economics Handbook, 1980; Schoenberg et al., 1982).

Propylene is expected to be released to the environment mainly in pyrolysis gases from the combustion processes and incidentally in gaseous emissions from industrial manufacturing and use operations. However, quantitative estimates of such industrial releases were not found. Sivertsen (1983) identified propylene as a constituent of the fugitive hydrocarbon emissions of an ethylene cracking unit at a petrochemical plant in Norway, but quantitative data were not presented.

Propylene is also expected to be released to the environment in the combustion gases of hydrocarbon fuels, wood, cigarettes, and synthetic polymers, refuse, etc. Jackson (1978) found that the propylene content of the exhaust gases of automobiles burning leaded and unleaded gasoline was lower in late-model cars equipped with oxidative catalytic converters (2.9 percent as carbon) than earlier models without converters (6.5 percent as carbon). Sigsby et al. (1987) found similar results with propylene content decreasing further to 1.9 percent as carbon in 1982 models. Propylene has also been detected in the exhaust gases of diesel-fuel-powered automobiles, emitted at a rate of 0.0207 g/mile (International Agency for Research on Cancer, 1979), and jet engines at maximum concentrations of about 130-143 ppm (Katzman and Libby, 1975). The compound has been found in the combustion gases and smoke from plant materials such as white pine wood (O'Mara, 1974) and cigarette tobacco treated with radiolabeled methoprene, an insect growth regulator (at 0.2 per cent of the total radioactivity recovered in the smoke) (Frisch et al., 1982). Levels of <0.4-1.5 ppm propylene were found in the flue gases of a municipal incinerator (Carotti and Kaiser, 1972).

Propylene can be released to the environment as emissions from incinerators burning hazardous waste. Allen et al. (1986) monitored stack emissions from an incinerator burning hazardous (infectious) hospital waste (infectious and noninfectious) and calculated an emission factor for propylene of <0.0011 g/kg. Propylene was not found above the detection limit (0.5 ppm) in any of 22 samples taken.

Thermal decomposition products of polymers such as polyethylene (Hoff et al., 1982), polyamides (Yoshida et al., 1978), and polyacrylonitrile (Woolley, 1982) have been found to contain propylene. Woolley reported that propylene was present at a concentration of about 10.7 ppm in the combustion gases following the burning of 5 kg of polypropylene in a 27-m^3 room (closed door). In addition to these sources, propylene is produced as a natural metabolite of trees and germinating beans, corn, cotton, and peas (Vancura and Stotzky, 1976; Kusy and Kusy, 1982).

Propylene concentrations in ambient air samples have been found to vary diurnally and with wind direction (Bos et al., 1977).

Sexton and Westberg (1984) reported that ground-level concentrations of propylene in urban air samples collected in seven U.S. cities ranged from 4 to 17 ppbC (geometric mean), whereas concentrations in rural surface air samples from six domestic sites ranged from <0.5 to 3.0 ppbC (geometric mean). Selected ambient air monitoring data are presented in Table 1-1.

Table 1-1. Propylene Concentrations in Selected Ambient Air Samples

Location	Concentration	Reference
Azusa, CA	4 ppb avg.	Altshuller et al. (1971)
Los Angeles, CA	10.5 ppb avg. 7-32 ppb (vol.)	Altshuller et al. (1971) Grosjean and Fung (1984)
Philadelphia, PA[a]	7-260 ppb	Giannovario et al. (1976)
Rural Southern Ontario	18-141 ppt	Anlauf et al. (1985)
Atlantic Ocean (marine air)	0.05 ppb	Rudolph et al. (1981)
Arctic air (spring haze)	1180 ppt avg. 5 ppt[b] 24 ppt	Schmidbauer and Oehme (1985) Khalil and Rasmussen (1984) Hov et al. (1984)
(summer)	187 ppt	Hov et al. (1984)
Bombay, India[a]	0.4-18.7 ppb 9.1 ppb (vol.) avg.	Netravalkar and Mohan Rao (1984) Mohan Rao and Pandit (1988)
Delft, The Netherlands	8 ppb max.	Bos et al. (1977)
Japan (industrial area near petrochemical plant)	10-100 ppb	International Agency for Research on Cancer (1979)
Jülich, FRG (semirural)	5 ppb	Rudolph et al. (1981)
Sydney, Australia	7.4 ppb (vol.) avg.	Nelson and Quigley (1982)
Lancaster, England	5.1 ppbc Geometric mean	Colbeck and Harrison (1985)
United Kingdom (rural)	0.7 ppb avg.; 0.10-4.8 ppb range	International Agency for Research on Cancer (1979)

[a]Suburban location near industrial complexes.
[b]Difference in concentration in and out of haze layers.

Propylene concentrations have been shown to be higher inside the home than outside. Lamb et al. (1985) detected propylene at concentrations of 0.6, 7.1, and 2.2 $\mu g/m^3$ inside a home in August 1981, December 1981, and June 1982, respectively. The ambient air concentrations outside the home were 0.9 and 0.5 $\mu g/m^3$ in December 1981 and June 1982, respectively. The authors attributed the increased levels of propylene (and other hydrocarbons) in the home to sources such as gas heating, automobile exhaust, and aerosol propellants.

Surface water samples taken from the Gulf of Mexico, Caribbean Sea, Atlantic Ocean, and Pacific Ocean have been found to contain

propylene at concentrations of 0.1-16 ng/l (Swinnerton and Lamontagne, 1974).

Estimates of occupational exposures to propylene have been reported in industrial hygiene surveys performed by the National Institute for Occupational Safety and Health. According to the National Occupational Hazard Survey (NOHS), 10,274 workers were potentially exposed to propylene in domestic workplace environments in 1970. Preliminary data for 1980 by the National Occupational Exposure Survey (NOES) indicate that 4,548 workers, including 136 women, were exposed to the compound.

Atmospheric concentration limits in the workplace have not been established for propylene by either the Occupational Safety and Health Administration or the American Conference of Governmental Industrial Hygienists. However, a suggested maximum permissible exposure limit of 4,000 ppm (6,984 mg/m^3) (one-fifth the lower flammable limit) has been suggested by Gerarde (1963).

Propylene has odor thresholds of 5.7-28.6 ppm (10-50 mg/m^3, detection) and about 57.3 ppm (100 mg/m^3, recognition), an organoleptic (taste) limit of 0.5 mg/l, and a light sensitivity to the eyes of approximately 6.3 ppm (11 mg/m^3) (Verschueren, 1983; Krasovitskaya and Malyarova, 1966).

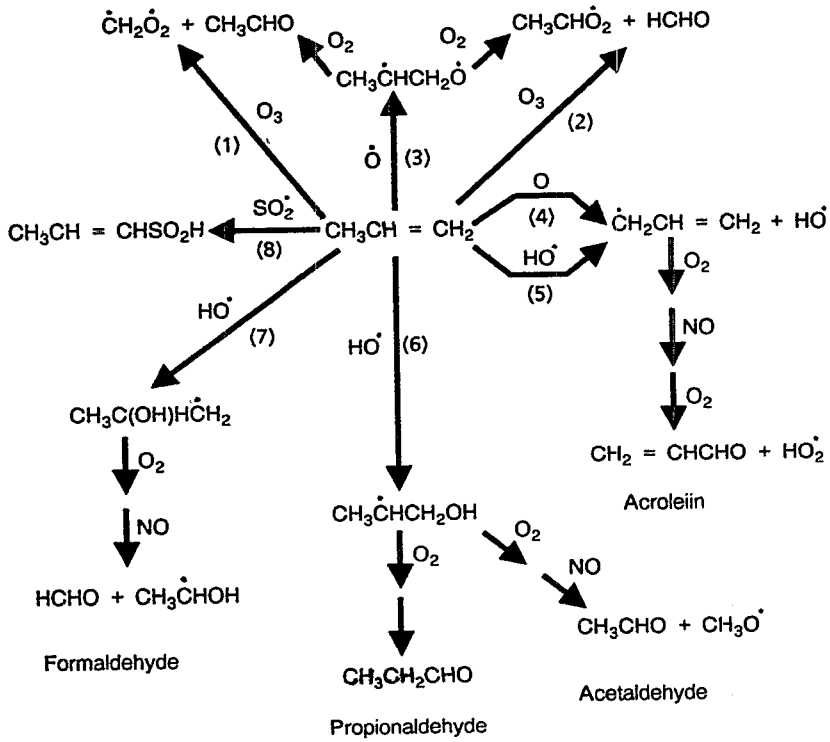
Propylene released to the environment from point and nonpoint sources is expected to partition into the atmosphere due to its high vapor pressure. However, its mobility may be limited because of the relatively higher density of propylene (1.45) as compared to air (1.0). Moreover, since propylene is also highly reactive, chemical reactions and degradative processes will probably limit its transport and decrease its residence time in the atmosphere.

The chemical properties of propylene are characterized by its double bond and allylic hydrogen atoms. The carbon-carbon double bond serves as a source of electrons and thus will easily undergo electrophilic addition reactions that are characteristic of alkenes. Several types of reactions that occur at room temperature include the addition of halogens and hydrogen halides, hydroxylation, glycol formation, and ozonolysis (Morrison and Boyd, 1973). Propylene reacts vigorously with oxidizing materials such as NO_2, N_2O_4, and N_2O. Furthermore, liquid propylene will explode on contact with water at 315-348 K (Schoenberg et al., 1982).

The atmospheric fate and photochemical reactions of propylene, such as the formation of photochemical oxidants and smog, have been studied by several investigators. Graedel et al. (1976) developed a mathematical model of photochemical processes to study the fundamental atmospheric chemistry of the troposphere. The model included chemical kinetics, time-related appearances of trace contaminated sources, solar flux variations, bulk airflow, and a geographical matrix of "reaction volumes." Data on the diurnal rates

of several groups of reactions between atmospheric components and propylene indicated that the principal reactant was the hydroxyl radical (OH), which accounted for more than 75 percent of the total propylene removed. The reactions of propylene with ozone occurred predominantly in the evening. The reactions of propylene with atomic oxygen or excited SO_2 were reported to be insignificant in the environment because of their relatively low concentration. Eight daylight photochemical reaction chains involved in the removal of propylene from the atmosphere are shown in Figure 1-1. Pathways 1, 2, and 3 produced two aldehyde molecules for each propylene molecule, whereas pathways 4-7 produced one aldehyde molecule and one free radical. The free radicals, in turn, produced additional hydrogen radicals by other reactions. The authors concluded that once the chain of propylene attack began, the production of aldehydes, organic radicals, and organic nitrate products was self-sustaining.

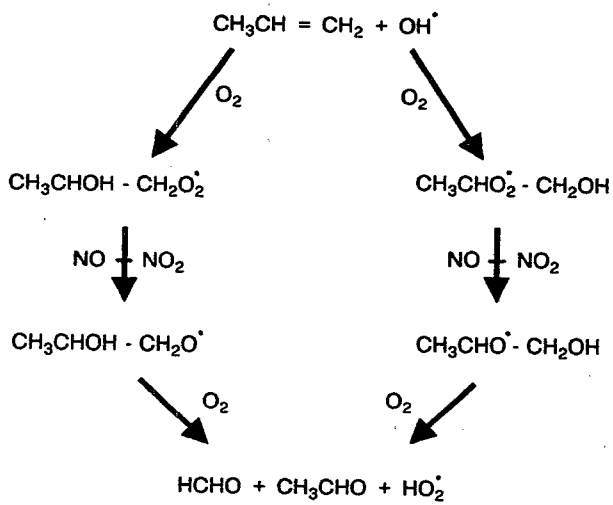
Figure 1-1. Daytime propylene reaction sequences in the urban troposphere. Source: Graedel *et al.* (1976).



The OH-initiated gas-phase oxidation of several compounds, including propylene, was studied by Edney et al. (1982). The

mechanism of this oxidation reaction with propylene is shown in Figure 1-2. Using the rate constant and average OH concentration values, the atmospheric half-life of propylene was calculated to be 7.7 hours at approximately 23°C.

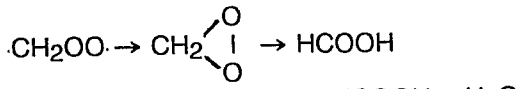
Figure 1-2. Hydroxyl radical-initiated gas-phase oxidation of propylene. Source: Edney *et al.* (1982).



The atmospheric residence time (T) of propylene can be estimated using the method of Lyman et al. (1982), which estimates residence time as a function of reaction rates with hydroxyl radicals and ozone but does not consider reactions with other substances or photodissociation. Therefore, T must be considered a maximum value. Using the rate constants for reactions of propylene with OH (k_{OH} = 1.5 x 10^{10} liter-mo1^{-1}-sec^{-1}) and O_3 (k_{O_3} = 7.8 x 10^3 liter-mol^{-1}-sec^{-1}), and average concentrations for OH (global annual average, 1.8 x 10^{-15} mol / l) and O_3 (urban annual average, 5.0 x 10^{-9} mol / l), the estimated T_{OH} is 10.3 hours and the T_{O_3} is 7.1 hours.

Akimoto et al. (1979, 1980) studied the photo-oxidation of propylene in an evacuable photochemical smog chamber. Quantitative analysis of the products was made *in situ* by a long-path Fourier transform infrared spectrometer. The products formed under dry or humid conditions (relative humidity 40 percent) in the photo-oxidation system containing propylene, NO_X, and air are listed in Table 1-2. Maximum ozone formation was obtained when the initial concentration ratio of propylene to NO_X was larger than 3. In the presence of water vapor, maximum ozone formation was slightly lower compared to the dry air system. There was no effect on the yield of other chemicals. However, the addition of water vapor markedly enhanced the yield of formic acid (from about 0.16 to 0.53

ppm maximum yield), which was ascribed to the reaction of propylene with formed ozone. The authors suggested that the mechanism may involve the following reactions:



$$\cdot CH_2OO\cdot \rightarrow CH_2\begin{matrix}O\\|\\O\end{matrix} \rightarrow HCOOH$$

$$\cdot CH_2OO\cdot + H_2O \rightarrow [CH_2OO\cdot H_2O] \rightarrow HCOOH + H_2O$$

Mutagenic, genotoxic, and cytotoxic effects of the photochemical reaction products of propylene plus NO_X have been reported. These are briefly discussed in Section 3.6.

The effects of water vapor and carbon monoxide on the photo-oxidation of propylene in the presence of NO_X were also investigated by Nguyen and Phillips (1979). Water vapor ($\leq$11,000 ppm) had no effect on the photo-oxidation rate of propylene by NO_X or on the yield of NO_2 and O_3. The formation of formic acid was not investigated by the authors. Carbon monoxide ($\leq$200 ppm), on the other hand, increased the rate of propylene photo-oxidation and product formation.

Table 1-2. Reactants and Products in the Propylene-NO_X-Air Photo-Oxidation System

	Initial Concentration, ppm				
Experiment	C_3H_6	NO_X	NO	NO_2	H_2O[a]
1	3.05	1.500	1.477	0.023	<1
2	3.04	1.403	1.358	0.045	1.7×104
3	3.06	1.583	0.016	1.567	<1
4	3.01	1.516	0.012	1.504	1.7×104

	Maximum Yield, ppm						
Experiment	O_3	HCHO	CH_3CHO	PAN[b]	PGDN[c]	N_2O_5	HNO_3
1	1.20	1.75	1.30	0.75	0.10	0.03	0.23
2	1.04	1.82	1.32	0.64	0.11	0.05	ND[d]
3	1.30	1.92	1.18	0.76	0.10	0.06	0.19
4	1.15	1.82	1.32	0.59	0.09	0.04	ND[d]

[a]RH ≈ 40% at 30°C.
[b]Peroxyacetyl nitrate.
[c]1,2-Propanediol dinitrate.
[d]Not determined due to H_2O interference.

Source: Akomoto et al. (1980).

Propylene glycol dinitrate was the major product in the gas-phase reaction of N_2O_5 with propylene in dry air in an evacuable smog chamber (Hoshino et al., 1978). Other products were acetaldehyde, propylene oxide, NO_2, and HNO_3. The ratio of propylene glycol dinitrate, acetaldehyde, and propylene oxide appeared to be independent of the N_2O_5 concentration. The authors suggested that the formation of propylene glycol dinitrate in the propylene-NO_x-air photo-oxidation system resulted from the reaction of propylene with generated N_2O_5 or NO_3 (nitric oxide) radical (Hoshino et al., 1978; Akimoto et al., 1979, 1980).

The gas-phase reactions of nitrate radicals with a series of organic compounds, including propylene, in air at room temperature were recently reported by Atkinson et al. (1984). The reactions with alkenes were shown to proceed via initial NO_3 radical addition to the double bond:

$$\cdot NO_3 + CH_3CH=CH_2 \rightarrow CH_3\overset{\overset{\displaystyle ONO_2}{|}}{C}HCH_2 + CH_3\,CHCH_2ONO_2$$

Under atmospheric conditions, the two radicals formed are expected to react with O_2 rapidly to yield the nitroxyperoxyalkyl nitrate and dinitrate as final products, although the exact reaction pathway for dinitrate formation is not known. The rate constant for the gas-phase reaction of propylene with nitrate radicals was reported to be $4.2 \pm 0.9 \times 10^{-12}$ liter-mol^{-1}-sec^{-1}. Based on the kinetic data generated in this study together with the observed ambient atmospheric concentrations of the NO_3 radicals that are more prevalent during the nighttime, the authors suggested that the reactions of the NO_3 radicals with the more reactive alkenes and hydroxy-substituted aromatic compounds may be an important nighttime sink for the radicals and the reactive organic compounds.

Shepson et al. (1985) conducted smog chamber studies of C_3H_6/N_2O_5 reactions at low (ppm) reaction levels to determine the various reaction mechanisms and yields for the production of organic nitrates. The concentration of N_2O_5 and propylene in the dark reaction were 0.68 and 1.85 ppm respectively. Major products positively identified were propylene glycol dinitrate, 2-hydroxypropyl nitrate, and 2-(nitrooxy) propyl alcohol. Limited evidence was also presented for the formation of α-(nitrooxy) acetone, which is produced by a series of reactions summarized as follows:

$$C_3H_6 + NO_3 \xrightarrow{O_2} CH_3C(O)CH_2ONO_2 \text{ (unbalanced)}$$

The authors reported that under their experimental conditions, which more closely approximate atmospheric conditions, the yields of propylene glycol dinitrate are not nearly or large as has been shown experimentally at higher initial concentrations of N_2O_5/C_3H_6. This was due to the competing reactions producing formaldehyde and acetaldehyde, or α-(nitrooxy) acetone. The formation of α-(nitro-

oxy) acetone was considered to be a significant finding, since it has been shown to be mutagenic (Shepson et al., 1985; Kleindienst et al., 1985; see also the mutagenicity section).

Upon release to surface waters, a small amount of propylene may dissolve in water, but most of the released material is expected to volatilize to the atmosphere. Propylene has been detected in marine and fresh waters, in trace amounts. Propylene surface levels in the Gulf of Mexico have been determined at 9.3×10^{-7}ml / l and in the North Atlantic at 5.9×10^{-7}ml / l water. Fresh water levels in Russia have been determined at 0.5 mg propylene in 1965 (Sandmeyer, 1985). In water bodies, propylene is readily degraded by microorganisms. Therefore, propylene is not expected to bioaccumulate or bioconcentrate in organisms and food chains. Under calm atmospheric conditions, it has been reported that propylene will sink to the ground because it is denser than air (OHMTADS). However, due to its high vapor pressure, it is expected that releases of propylene to soil would be readily transported to air or water.

Two studies were reported on the microbial oxidation and metabolism of propylene (Cerniglia et al., 1976; Perry, 1980). Propane-grown cells of *Mycobacterium convolutum* oxidized propylene rapidly to acrylate. The rate of oxidation was 51 µl O_2/mg cells/hour. However, microbial growth was not observed because the acrylate inhibited fatty acid oxidation by these microorganisms. A mixed culture of two microbial species capable of utilizing propylene as the sole source of carbon and energy was also isolated by soil enrichment (Cerniglia et al., 1976). One was a red-pigmented organism designated YS-3R and identified as being similar to *Thermus aquaticus* (a thermophilic heterotrophic organism), and the other was a small, white, colony-forming organism designated YS-3 (Perry, 1980). An axenic culture isolated from marine soil and designated as strain PL-1 also metabolized propylene and utilized it as the sole carbon source. The rate of oxidation was 81 µl O_2/mg cells/hour. Radiolabeled experiments with [$1\text{-}^{14}C$] propylene and $^{14}CO_2$ in the presence or absence of arsenite (an inhibitor of pyruvate metabolism) indicated that propylene was not metabolized into pyruvate, but rather was cleaved into two products, the first containing one carbon atom and the second containing two. Isocitrate lyase activity and fatty acid profile determination further indicated that the mixed culture and strain PL-1 oxidized propylene at the double bond, resulting in cleavage of the molecule.

In contrast, the ratio of propylene's 5-day biochemical oxygen demand to its chemical oxygen demand (5-day BOD/COD), used as an estimate of biodegradability, was zero, indicating that it was relatively undegradable (Lyman et al., 1982). However, the experimental conditions for BOD determination were not presented, and a complete evaluation of this finding could not be made.

1.3 Environmental Effects

The available literature on the ecological effects of propylene was very limited. Data were found only on the *in vivo* and *in vitro* effects of propylene on higher plants. No toxicity data were found on the effects of propylene on aquatic plants, invertebrates, microorganisms, or fish. Van Haut and Prinz (1979) reported that propylene's toxicity to plants was essentially insignificant when compared to ethylene, and they concluded that propylene emissions would have practically no adverse effects on plant growth. In a short-term test, plants, including the bushbean, radish, cress, petunia, and geranium, showed characteristic damage following exposure to ethylene at concentrations ranging from 0.1 to 1.44 mg/m^3 for varied periods of time; i.e., 0.5-14.0 days. Propylene concentrations ranging from 360 to 1500 times those of ethylene (actual concentrations not given) were needed to achieve comparable growth inhibition, but such high propylene concentrations are not expected to be found in the ambient air.

In an *in vitro* study of the mechanism of action of ethylene as a plant growth inhibitor, Smith et al. (1983) examined the effect of ethylene and some of its analogs, including propylene, on the oxidation of indole-3-acetic acid. Ethylene and its analogs inhibited the oxidation of indole-3-acetic acid by peroxidase under conditions where the $Fe_p + + \leftrightarrows$ Compound III (an oxy-ferrous complex of peroxidase) shuttle was activated. Inhibition occurred only in the presence of the superoxide anion radical (O_2^-). Spectral and kinetic data indicated that ethylene and its analogs enhanced the rate of reaction of O_2^- with peroxidase; i.e., the $Fe_p + + \leftrightarrows$ Compound III shuttle, resulting in the formation of Compound III. Propylene was a less effective inhibitor than ethylene.

2. HEALTH EFFECTS

2.1 Pharmacokinetics and Metabolism

Limited information was available on the metabolism of propylene. The kinetics of uptake and metabolism of propylene in male CBA mice was studied by Svensson and Osterman-Golkar (1984). Groups of 15 mice were placed in a closed recirculation 11-liter chamber and exposed to propylene at eight concentrations ranging from 95 to 1,715 ppm. The level of propylene in the air was determined by gas chromatography and was followed for at least one half-life during the exposure period. The rate of uptake at equilibrium (between 100 and 150 minutes after onset of exposure) was estimated from the slope of the concentration-time curve and expressed in milligrams of propylene per kilogram of body weight per hour. The metabolism of propylene followed Michaelis-Menton kinetics. A double reciprocal plot of these rates versus chamber concentration (which is a Lineweaver-Burk representation of Michaelis-Menton kinetics) allowed determination of the K_m and V_{max}. The K_m (concentration in air at which uptake proceeds at half-maximum velocity) was 800 ± 60 ppm, and the V_{max} (maximum rate of uptake) was 8 ± 0.5 mg/kg/hour. Since propylene was expected to be oxidized to propylene oxide in a manner similar to that previously demonstrated for ethylene in mice, the combined uptake of both ethylene and propylene was studied. It was found that the uptake of ^{14}C-ethylene (0.01 ppm), in the presence of a high concentration of propylene (1,260 ppm), was lower than in the absence of propylene. This finding suggested a competitive interaction of both compounds in their metabolic pathways and indicated that propylene is converted *in vivo* to propylene oxide. The authors reported that the metabolic pathway of propylene involved oxidation to the corresponding oxide and subsequent alkylation of nucleophilic sites in macromolecules. Alternatively, propylene and/or propylene oxide were metabolized by other pathways and excreted (see also below).

2.2 Biochemical Effects

Svensson and Osterman-Golkar (1984) also studied the alkylation of macromolecules in mice exposed to ^{14}C-propylene to demonstrate the transient appearance of propylene oxide in tissues. In one experiment, the mice were exposed for 1 hour to an atmosphere containing 2830 ppm of ^{14}C-propylene and then sacrificed 13 hours later. Hemoglobin from blood and DNA from the

liver, testes, spleen, lungs, and kidneys were isolated, and labeled alkylation products in these macromolecules were identified. The amount of radioactivity bound to the DNA of the liver and pooled organs was below the level of detection. The hemoglobin was hydrolyzed, and the resulting amino acids chromatographed by ion exchange after adding carrier amino acids and 2-hydroxypropylated derivatives of cysteine, histidine, and valine. The major part of the radioactivity of hemoglobin (about 70 percent) eluted with serine, glycine, and alanine, indicating extensive biodegradation of propylene followed by biosynthetic incorporation of 1 and 2 ^{14}C-carbon fragments into amino acids. Hemoglobin hydrolysates also showed radioactivity associated with S-(2-hydroxypropyl) cysteine, N-(2-hydroxypropyl) valine, and N-(2-hydroxypropyl) histidine. The degree of alkylation of cysteine and valine was 2 nmol/g of hemoglobin, and that for histidine was 0.9 nmol/g of hemoglobin. In another experiment, mice were exposed to 20,000 ppm of unlabeled propylene for 4 hours/day for 8 days. Blood was collected immediately after the last exposure and hemoglobin alkylation assessed. The degree of alkylation was 2.2 nmol N-(2-hydroxypropyl) histidine/g of hemoglobin per hour. Two diastereoisomers of the histidine derivative were isolated, which were similar to those obtained from the reaction of DL-propylene oxide with histidine. The authors suggested that these results indicate nonstereospecific oxidation of propylene to propylene oxide. However, the data presented do not necessarily support such a mechanism.

The role of the cytochrome P-450 system in the hepatotoxicity of propylene was investigated by Osimitz and Conolly (1985). Male Sprague-Dawley rats (200- 275 g) were pretreated with either polychlorinated biphenyls (PCB, Aroclor 1254), phenobarbital (PB), β-naphthoflavone (BNF), or BNF + PB to maximally induce the appropriate isozymes of cytochrome P-450. Appropriate vehicle control groups were also included. The animals (at least four rats per group) were then exposed to CP-grade propylene (>99.5 percent pure) at 0 or 50,000 ppm for 4 hours. All animals were sacrificed 20 hours postexposure, and measurements of serum sorbitol dehydrogenase (SDH) and alanine leucine transaminase (ALT) activities were taken as indicators of hepatotoxicity. Propylene was not hepatotoxic in untreated, PB-, BNF-, or BNF + PB-treated animals, but it was hepatotoxic in rats pretreated with PCB. The authors concluded that an isozyme of cytochrome P-450 induced by PCB is required to convert propylene to a hepatotoxic metabolite in pretreated rats.

2.3 Acute Toxicity

No LD_{50} values or lethal dose estimates were found in the literature. Reports in the early literature indicate that propylene is not very toxic. Exposure of white mice to a mixture of 50 percent propylene and 50 percent air produced anesthesia in 8 minutes (Riggs, 1925). Riggs (1925) performed a study in which white rats of

a "pure strain and great uniformity" were exposed to a gas mixture containing propylene (purity, 99 percent) at concentrations ranging from 40 to 70 percent. Oxygen levels were maintained at 25 percent, whereas the concentrations of nitrogen were varied. The sex and number of animals exposed were not reported. At 40 percent, propylene produced a light anesthesia within 15-20 minutes; exposure up to 6 hours produced no visible signs of toxicity. Exposure to propylene at 55, 65, 70 percent produced a deep anesthesia within 3-6, 2-5, 1-3 minutes, respectively. Animals exposed to 55 percent propylene for 7.5 hours recovered within 2 minutes postexposure and exhibited no signs of overt toxicity. However, propylene concentrations of 65 or 70 percent led to respiratory failure and death after 2 hours or 20-25 minutes, respectively.

Brown (1924) reported similar effects in cats. Anesthesia was induced and maintained at propylene concentrations between 37 and 50 percent. At propylene concentrations of 65 and 70 percent, a fall in blood pressure was reported. In dogs, propylene has been shown to be a cardiac sensitizer (Krantz et al., 1948). Two mongrel dogs were administered intravenous injections of epinephrine hydrochloride at 0.01 mg/kg and then allowed to breathe a propylene/air mixture (propylene concentration not specified) for 10 minutes. Propylene caused a sensitization of the myocardium to the epinephrine in both dogs, resulting in multifocal ventricular tachycardia that progressed to ventricular fibrillation and caused death.

No further information was found on cardiac sensitization to epinephrine following propylene exposure.

2.4 Subchronic Toxicity

No compound-related toxic effects were observed in male and female F344/N rats and $B6C3F_1$ mice following repeated exposure to propylene for 14 days (5 animals/sex/dose) or 14 weeks (10 animals/sex/dose) at concentrations of 0, 625, 1,250, 2,500, 5,000, and 10,000 ppm for 6 hours/day, 5 days/week (National Toxicology Program, 1985). In an early study (Reynolds, 1926), six white mice of unspecified strain and sex were exposed to impure propylene at a concentration of 35 percent for five exposures of 90 minutes each over a 10-day period. No toxic effects were noted except for the liver of one animal, which showed moderate fatty degeneration. Subchronic exposure of single mice for 60 minutes (11 exposures in 33 days, 14 exposures in 41 days, and 20 exposures in 58 days) did not cause any obvious toxic effects except for slight fatty degeneration of the livers of the mice receiving 14 and 20 exposures.

2.5 Chronic Toxicity

Quest et al. (1984) recently reported the results of an NTP carcinogenicity/chronic toxicity bioassay with propylene in F344/N rats and $B6C3F_1$ mice. Groups of 50 rats and mice of each sex were

exposed to air containing propylene at concentrations of 5,000 or 10,000 ppm for 6 hours/day, 5 days/week for 103 weeks. Control groups were exposed in the same manner but to clean, filtered air. For safety reasons, the highest dose level used was based on a concentration that was one-half the lower flammability limit of propylene, and may have been below the maximum tolerated dose. Propylene had no effect on weight gain or mortality when compared to controls in either species or sex.

Histopathologic findings indicated a positive trend in the incidence of C-cell hyperplasia in the thyroid glands of propylene-exposed rats. In addition, increased incidences of nonneoplastic lesions developed in the nasal cavities of the exposed rats (Table 2-1). These lesions included epithelial hyperplasia in the anterior part of the nasal cavity in females exposed at 10,000 ppm; squamous metaplasia in males and females exposed at 5,000 ppm and in females exposed at 10,000 ppm; and inflammatory changes, characterized by an influx of lymphocytes, macrophages, and granulocytes, in the nasal lumen of males exposed at both concentration levels.

Table 2-1. Incidence of Epithelial Changes in the Nasal Cavities of Rats Exposed to Propylene via Inhalation

	Exposure level		
Observation	0	5,000 ppm	10,000 ppm
Epithelial hyperplasia			
Male	2/50 (4%)	2/50 (4%)	5/50 (10%)
Female	0/49 (0%)	4/50 (8%)	9/50 (18%)[a]
Squamous metaplasia			
Male	2/50 (4%)	19/50 (38%)[a]	7/50 (14%)
Female	0/49 (0%)	15/50 (30%)[a]	6/50 (12%)[a]
Inflammation			
Male	11/50 (22%)	21/50 (42%)[a]	19/50 (38%)
Female	8/49 (16%)	10/50 (20%)	13/50 (26%)

[a]Significantly ($p < 0.05$) higher than control values.

Source: Quest et al. (1984).

There were no corresponding lesions of the nasal cavities in mice exposed to propylene. However, there was an increased incidence of chronic, focal, renal inflammation in males (0 of 50, 17 of 49, and 9 of 49) and in females (1 of 50, 7 of 49, and 6 of 49) exposed to propylene at 0, 5,000, and 10,000 ppm, respectively.

2.6 Mutagenicity

Two studies have evaluated the mutagenic potential of propylene. The compound was nonmutagenic in an *E. coli* survival assay and the *Salmonella*/microsomal reverse mutation assay. Landry and Fuerst (1968) reported that propylene was not mutagenic to *E. coli* B. The pure gas was bubbled through a 10-ml suspension of the cells for 10 minutes and the cells subsequently maintained under the

gaseous atmosphere for 24 hours. Mutation rates were determined by counting the survival cells, which formed colonies after treatment with penicillin and neutralization with penicillinase.

Propylene was also evaluated for mutagenicity in the Ames reversion assay and found to be nonmutagenic in *Salmonella typhimurium* tester strains TA97, TA98, and TA100 (Hughes et al., 1984). The compound, diluted in ethanol, was tested at dose levels ranging from 6.3 to 626.0 µg/plate, in triplicate, with and without metabolic activation. Metabolic activation consisted of two liver S9 systems (rat and hamster) induced with Aroclor 1254.

Recently, two reports have described the mutagenic, genotoxic, and cytotoxic effects of the photochemical reaction products of propylene and NO_x. The photooxidation products of C_3H_6/NO_x showed increased mutagenic activity when compared to the reactants in the Ames assay using *Salmonella typhimurium* tester strain TA100 (Kleindienst et al., 1985). The tester strain was exposed for 20 hours to the irradiated products of the reactants C_3H_6, NO, and NO_x-NO at initial concentrations of 826, 505, and 200 ppb, respectively. Metabolic activation (S9 from livers of male CD-1 rats induced with Aroclor 1254) had no significant effect on mutagenic activity; however, the number of revertants did increase as a function of exposure time (~160 excess revertants/plate after 2.7 hours and ~625 revertants/plate after 7.5 hours of exposure). An attempt was made to account for the excess revertants by summing the contributions of the individual major and minor reaction products (the major reaction products of this system are described in Section 1.2). Of the major and minor reaction products, only HCHO and PAN had mutagenic activities above the detection limit, contributing 19 and 27 revertants/plate, respectively, after 7.5 hours of exposure. When the number of revertants contributed by minor products, such as peroxide, was added to those contributed by PAN and HCHO, only 20 percent of the observed mutagenic response could be accounted for. Another possible product of the photochemical reaction of propylene and N_2O_5 suggested by Shepson et al. (1985) is α-(nitrooxy) acetone which has been shown to be mutagenic.

S. typhimurium strain TA100 was also exposed to the products of the C_3H_6/N_2O_5 dark reaction to determine to what extent C_3H_6/NO_3 reactions contributed to the mutagenic response. A small mutagenic response was observed for this mixture (180 excess revertants/plate above controls), with S9 activation having no significant effect on mutagenic activity. The contribution to the total response for reaction products α-nitratoacetone, HCHO, and PAN was estimated to be 22, 2, and 8 revertants/plate, respectively. However, the contribution of all known reaction products accounted for only a small fraction of the total observed response (~19 percent). A number of possible explanations for the observed mutagenic effect were suggested by the authors, including the presence of an unknown potent mutagen not detected in the analysis

of reaction products, the production of a mutagen from a reaction that occurred in the test medium, and the combined effect of two or more reaction products acting synergistically (Kleindienst et al., 1985).

The photochemical reaction products of propylene and NO_2 increased the frequency of sister-chromatid exchanges (SCE's) and the rate of growth inhibition in cultured Chinese hamster V79 cells (Shiraishi and Bandow, 1985). Cultured cells were exposed for 2 hours to the photochemical reaction products produced in a smog chamber using four initial concentration ratios of reactants (propylene: NO_2): 0.5:0.25, 1.0:0.5, 2.0:1.0, and 3.0:1.5 ppm. At the lowest concentration of reaction products, the mean SCE frequency was significantly higher when compared to control cells (14.18 per cell versus 6.84 per cell, $p < 0.001$). At higher concentrations, a greater frequency of SCE was observed, approximately two to four times greater than control cells (significantly different at $p < 0.001$). The photochemical reaction products inhibited cell growth in a dose-dependent manner, with a mean inhibition rate of 7, 22, 44, and 59 percent, respectively, for the four levels of concentrations tested. In contrast, propylene and NO_2 alone failed to induce SCE's in V79 cultures.

Cell cultures were also exposed for 2 hours to NO_2 and O_3 concentrations ranging from 0.5 to 8.0 and 0.13 to 1.0 ppm, respectively. Both compounds significantly increased the frequency of SCE's in V79 cells, but their effects were weaker than those induced by the photochemical reaction products of propylene and NO_2. The authors concluded that the strong induction of SCE's in V79 cells by the photochemical reaction products was due to a multiple effect resulting from an interaction between reaction products (Shiraishi and Bandow, 1985).

One study reports the mutagenic effects of the reaction products of O_3 and propylene on *E. coli* K 12 343/113. Nover and Botzenhart (1985) showed induction of gal^+, arg^+ and MTR-mutations in growing cells after treatment with ozonized propylene. The treatment with ozone was carried out under UV irradiation (3000-4000 angstroms) to increase the concentration of the reaction products which were hydrocarbon radicals, peroxides, aldehydes and ketones. Similar reaction products of propylene with O_3 are shown in Figure 1-1. Nover and Botzenhart (1985) relate the mutagenic effects to the "photochemical smog" reaction products but not to propylene or other hydrocarbons studied.

2.7 Carcinogenicity

Two studies have been reported on the carcinogenic potential of propylene in rats and mice. In one, Maltoni et al. (1982) presented the results of carcinogenicity studies with several chemicals, including propylene, conducted in an attempt to identify potential brain carcinogens. In the study with propylene, groups of 120

Sprague-Dawley rats were exposed to propylene in air at 200, 1,000, or 5,000 ppm for 7 hours/day, 5 days/week for 104 weeks. One female rat exposed at 1,000 ppm had a meningioma. However, since 2 of the total 325 female controls in the study also had meningiomas, the authors concluded that propylene did not cause brain tumors in rats. No data were presented for any other parameters of the study.

The more definitive study was the NTP carcinogenicity/chronic toxicity bioassay with propylene in F344/N rats and $B6C3F_1$ mice reported by Quest et al. (1984). There was no increase noted in the incidence of any neoplasm in the propylene-exposed rats when compared with the controls. In fact, a negative ($p < 0.05$) trend was observed in the incidence of total thyroid tumors in female rats, with the incidence of C-cell adenomas of the thyroid in the 10,000-ppm group significantly ($p < 0.05$) lower than in controls. However, as indicated above, there was a positive trend in the incidence of C-cell hyperplasia in female rats.

In the exposed mice, both increases and decreases were noted in the incidences of certain tumors (Table 2-2). In female mice, significant ($p < 0.05$) positive trends in the incidences of hemangiosarcomas, combined hemangiomas and hemangiosarcomas, and uterine endometrial stromal polyps were observed in the high dose group, but the incidences were not significantly different from controls. Similarly, in exposed males, significant ($p < 0.05$) decreases in the incidences of hepatocellular adenomas, lung adenomas, and combined lung adenomas and carcinomas were observed in the low dose group although this was not seen in the high dose group. There was, however, a significant ($p < 0.05$) negative trend in the incidence of lung adenomas and carcinomas in males, but the biological meaning of this is questionable, since the matched control rate was considerably higher than the historical control rate.

Table 2-2. Incidence of Tumors in Mice Exposed to Propylene via Inhalation[a]

	Exposure level, ppm		
	0	5,000	10,000
Females			
Hemangiosarcomas	0/50	0/49	3/50
Hemangiomas and hemangiosarcomas	0/50	1/49	4/50
Uterine endometrial stromal polyps	0/47	0/47	3/48
Males			
Hepatocellular adenomas	5/50	0/49	3/49
Lung adenomas	9/50	1/49	4/50
Lung adenomas and carcinomas	16/50	4/49	7/50[b]

[a]Only neoplasms with incidences apparently different from controls.
[b]Significant ($p < 0.05$) negative trend.

Source: Quest et al. (1984).

Under the conditions of the study and at the concentrations tested, it was concluded that propylene is not carcinogenic in F344/N rats and $B6C3F_1$ mice.

In addition to these two studies, Quest et al. (1984) reported that in a preliminary report by Maltoni, no carcinogenic response was found in Swiss mice exposed by inhalation for 18 months, to propylene at concentrations of 200, 1000, or 5000 ppm for 7 hours/day, 5 days/week.

A weight of evidence characterization for propylene using EPA's Guidelines for Carcinogen Risk Assessment involves taking account of several types of information:

- The NTP bioassays in rats and mice provide some indication of carcinogenic activity, such as positive trends in the incidence of hemangiosarcomas and combined hemangiomas and hemangiosarcomas in female mice and a hint of bladder hyperplasia in female rats.
- Propylene is metabolized to propylene oxide (the strength of this data base requires further scrutiny). Propylene oxide and ethylene oxide are alkylating agents, and there is sufficient animal evidence for their carcinogenicity.
- The similarity of endpoints in the mouse between propylene (trend of hemangiomas) and propylene oxide (incidence of hemangiomas) provides additional indication of potential carcinogenic activity.

Since no epidemiological data are available, the human data base is inadequate. The available direct animal bioassay data for propylene can be viewed as inadequate but bordering on limited. The animal evidence is elevated to the limited category by considering the supporting information on metabolites to include the carcinogenicity of metabolites and the commonality of site of action between the metabolites and parent compound propylene. Thus the overall weight-of-evidence becomes Group C, possibly a human carcinogen.

A quantitative cancer unit risk derivation is not warranted at this time, given that there is not a statistically significant animal dose-response study available, and thus the quantitative analysis cannot be developed.

2.8 Other Health Effects

No studies were found on teratogenicity, reproductive effects, neurotoxicity, or other types of health effects on humans.

3. SUMMARY

Propylene is an olefinic hydrocarbon with the chemical structure $CH_3CH=CH_2$. It is highly soluble in water and has a high vapor pressure. Current domestic production capacity has been estimated to be about 22 billion pounds. Propylene is a product obtained from crude oil by distillation and used in several chemical and refinery processes. In the chemical industry, propylene is used as a starting material in the manufacture of polypropylene, acrylonitrile, propylene oxide, and as a component in fuel. In refinery applications, it is used as a feedstock or additive to gasoline and fuels.

Propylene may be released to the environment in gaseous emissions from industrial production, chemical utilization, and various combustion processes. Ambient concentrations in urban areas of the United States have generally been found to range from about 1 to 10 ppb, whereas concentrations in rural air samples are approximately an order of magnitude lower.

Gas chromatography with flame ionization detection and mass spectroscopy has been used extensively to measure concentrations of propylene in environmental media.

Propylene released to the environment from point and nonpoint sources is expected to partition into the atmosphere due to its high vapor pressure. However, propylene has a relatively higher density and is also highly reactive; therefore, its transport will be limited.

Propylene is one of several "volatile organic compounds" that are precursors of photochemical oxidants and other smog components. In ambient air, propylene is susceptible to attack by hydroxyl radicals and ozone, although the former accounts for more than 75 percent of the total propylene removed. The reactions of propylene with hydroxyl radicals occur mainly during the daytime; with ozone, during the evening; and with NO_3 radicals, at night. The end products of these reactions are several aldehydes (e.g., acrolein, formaldehyde, and acetaldehyde), acids, ketones, and nitrates, but other products have also been identified. The atmospheric half-life of propylene has been estimated to be 7.7 hours.

Trace levels of propylene have been detected in marine and fresh waters. In water, propylene would probably be subjected to chemical and degradative processes. Propylene is not expected to bioconcentrate or bioaccumulate in organisms and food chains, and has been reported to be metabolized by some microorganisms. Available environmental effects data are limited to studies with higher

plants. Some toxic effects were reported at very high concentrations; however, these concentrations are not expected to be found in the environment, except for cases associated with industrial leaks or spills.

One study on the uptake and metabolism of propylene in mice following inhalation exposure indicates that propylene is readily metabolized, having K_m and V_{max} values of 800 ± 60 ppm and 8 ± 0.5 mg/kg/hour, respectively. Propylene is apparently oxidized to propylene oxide and also metabolized into 1 and 2 carbon fragments, which are readily incorporated into certain amino acids. Propylene and/or propylene oxide also alkylates blood hemoglobin in mice via conjugation with cysteine, valine, or histidine to form the corresponding S- and N-(2-hydroxypropyl) derivatives.

Limited data from acute toxicity studies indicate that propylene is not very toxic to rats (strain and sex not reported); only light anesthesia was noted following exposure to a 40 percent concentration of propylene for 6 hours. Similarly, no compound-related toxic effects were found in subchronic studies with F344/N rats and $B6C3F_1$ mice exposed to propylene at concentrations up to 10,000 ppm for 6 hours/day, 5 days/week for 14 days or 14 weeks. Propylene was also not carcinogenic in F344/N rats and $B6C3F_1$ mice following exposure at concentrations up to 10,000 ppm for 6 hours/day, 5 days/week for 103 weeks. However, nonneoplastic lesions such as inflammation, epithelial hyperplasia, and squamous metaplasia developed in the nasal cavities of exposed rats, and renal inflammation was noted in exposed mice.

Propylene was not mutagenic in an *Escherichia coli* assay when bubbled through the cells for 10 minutes, nor in *Salmonella typhimurium* strains TA97, TA98, and TA100 at dose levels up to 626 µg in ethanol per plate. Mutagenic and cytotoxic effects of photochemical reaction products of propylene and NO_x have recently been reported in *S. typhimurium* strain TA100 and cultured Chinese hamster V79 cells without activation; mutagenic effects of the reaction products of O_3 and propylene on *E. coli* K12 343/113 have also been reported; no other mutagenicity studies were available. No teratogenicity, reproductive effects, or neurotoxicity studies of propylene with laboratory animals were found in the available literature. Similarly, there were no epidemiologic or case studies available to assess its effects on humans.

According to EPA's carcinogen risk assessment guidelines, the animal evidence for the carcinogenicity of propylene is judged to be only limited. This equates to a Group C classification, given that there is no epidemiologic data and the animal evidence is limited.

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