UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
WASHINGTON, D.C. 20460

NOV 12 1991

MEMORANDUM

OFFICE OF
PESTICIDES AND TOXIC
SUBSTANCES

Subject: Supplemental Response to Re-registration Phase 4 for Propylene Oxide. 6(a)(2) Data. Record # S404645
EPA ID # 042501

To: Betty Crompton/Barbara Briscoe PM # 51 Tox Chem # 713A
Accelerated Reregistration Branch Proj. No 2-0253
Special Review and Reregistration Division (H7508W)

From: Joycelyn E. Stewart, Ph.D., Acting Head, 11/5/91
Section 2, Toxicology Branch I,
Health Effects Division (H7509C)

KB 11/5/91

Thru: Stanley Gross, Ph.D.
Section 2, Toxicology Branch I
Health Effects Division (H7509C) 11/6/91

Action Requested: Review 28 month inhalation study in rats designated 6(a)(2).

Conclusion: Toxicology Branch I acknowleges the submission by Science Regulatory Services International of a 28 month chronic/oncogenicity inhalation study in SPF Wistar rats on behalf of Aberco, Inc., to support the re-registration of Propylene Oxide.

The information contained in this submission is included in the IRIS data base on Propylene Oxide. Based on these data and additional information reviewed, the chemical has been classified by CRAVE as class B2, a probable human carcinogen. It is unlikely that the submitted information will adversely affect the manner in which the chemical will be regulated. The inhalation study will be placed in the normal queue for review.

Propylene oxide; CASRN 75-56-9 (08/01/91)

Health risk assessment information on a chemical is included in IRIS only after a comprehensive review of chronic toxicity data by work groups composed of U.S. EPA scientists from several Program Offices. The summaries presented in Sections I and II represent a consensus reached in the review process. The other sections contain U.S. EPA information which is specific to a particular EPA program and has been subject to review procedures prescribed by that Program Office. The regulatory actions in Section IV may not be based on the most current risk assessment, or may be based on a current, but unreviewed, risk assessment, and may take into account factors other than health effects (e.g., treatment technology). When considering the use of regulatory action data for a particular situation, note the date of the regulatory action, the date of the most recent risk assessment relating to that action, and whether technological factors were considered. Background information and explanations of the methods used to derive the values given in IRIS are provided in the five Background Documents in Service Code 5, which correspond to Sections I through V of the chemical files.

STATUS OF DATA FOR Propylene oxide

File On-Line 10/01/90

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	no data	
Inhalation RfC Assessment (I.B.)	on-line	11/01/90
Carcinogenicity Assessment (II.)	on-line	02/01/91
Drinking Water Health Advisories (III.A.)	no data	
U.S. EPA Regulatory Actions (IV.)	no data	
Supplementary Data (V.)	no data	

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I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Propylene oxide
CASRN -- 75-56-9

Not available at this time.

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

Substance Name -- Propylene oxide
CASRN -- 75-56-9
Last Revised -- 11/01/90

The inhalation Reference Concentration (RfC) is analogous to the oral RfD and is likewise based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis, but may not exist for other toxic effects such as carcinogenicity. The inhalation RfC considers toxic effects for both the respiratory system (portal-of-entry) and for effects peripheral to the respiratory system (extrarespiratory effects). It is appropriately expressed in units of mg/cu.m. In general, the RfC is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily inhalation exposure of the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime. Inhalation RfCs are derived according to the Interim Methods for Development of Inhalation Reference Doses (EPA/600/8-88/066F August 1989) developed by U.S. EPA scientists and peer-reviewed. For more information on the interim nature of these methods and future plans see the INFO. Section of IRIS. RfCs can also be derived for the noncarcinogenic health effects of compounds which are carcinogens. Therefore, it is essential to refer to other sources of information concerning the carcinogenicity of this substance. If the U.S. EPA has evaluated this substance for potential human carcinogenicity, a summary of that evaluation will be contained in Section II of this file when a review of that evaluation is completed.

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___I.B.1. INHALATION RfC SUMMARY

Critical Effect	Exposures*	UF	MF	RfC
Nest-like infolds of the nasal respiratory epithelium	NOAEL: None	100	1	3E-2 mg/cu.m
	LOAEL: 71 mg/cu.m (30 ppm)			
	LOAEL(ADJ): 13 mg/cu.m			
2-Year Rat Chronic Inhalation Study	LOAEL(HEC): 2.9 mg/cu.m			
Kuper et al., 1988				

*Conversion Factors: MW = 58.08. Assuming 25C and 760 mmHg, LOAEL (mg/cu.m) = 30 ppm x 58.08/24.45 = 71. LOAEL(ADJ) = 71 mg/cu.m x 6 hours/day, 5 days/week = 13. The LOAEL(HEC) was calculated for a gas:respiratory effect in the ExtraThoracic region. MVa = 0.30 cu.m/day, MVh = 20 cu.m/day, Sa(ET) = 11.6 sq. cm, Sh(ET) = 177 sq. cm. RGDR(ET) = (MVa/Sa) / (MVh/Sh) = 0.23. LOAEL(HEC) = LOAEL(ADJ) x RGDR = 2.9 mg/cu.m.

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___I.B.2. PRINCIPAL AND SUPPORTING STUDIES (INHALATION RfC)

Kuper, C.F., P.G.J. Reuzel, V.J. Feron and H. Verschuuren. 1988. Chronic inhalation toxicity and carcinogenicity study of propylene oxide in Wistar rats. Food Chem. Toxicol. 26(2): 159-167.

The nasal mucosa was the primary tissue affected by exposure to propylene oxide in a chronic inhalation toxicity study conducted by Kuper et al. (1988). One-hundred Wistar rats/sex/group were exposed to 0, 30, 100, or 300 ppm

propylene oxide (0, 71, 238, and 713 mg/cu.m) 6 hours/day, 5 days/week for 123 weeks (females) and for 124 weeks (males, duration adjusted concentrations: 0, 13, 43, and 127 mg/cu.m). Interim sacrifices were performed on 10 rats/sex/group after 12, 18, and 24 months of exposure with remaining animals sacrificed at 28 months. Hematology, urinalysis, serum chemistry, and histopathology, including lung, trachea, bronchial lymph nodes, spinal cord, and skeletal muscle, were performed. Survival was adversely affected by exposure to propylene oxide; by week 115, there was a statistically significant increase in mortality in rats of both sexes exposed to 300 ppm propylene oxide (HEC = 127 mg/cu.m, based on an extrarespiratory effect assuming periodicity). By week 119, mortality was significantly increased (43% compared with 30% in controls) in the female rats exposed to 100 ppm (43 mg/cu.m), but not at other times during the study. A FEL is identified at 300 ppm (FEL[HEC] = 127 mg/cu.m). Body weights were statistically significantly reduced in the high-dose males throughout the study, but in females the weight reduction was only significant in the high-dose group for the first year of the study. Body weight data were not included in the report. The NOAEL for body weight changes is 100 ppm [NOAEL(HEC) = 43 mg/cu.m]. The authors state that no treatment-related changes were observed in any of the biochemical, urinalysis, or organ weight endpoints but detailed data were not presented. Statistically significantly increased incidences of several non-neoplastic degenerative and hyperplastic nasal lesions were observed in all exposure groups. These changes occurred in the respiratory and olfactory epithelium of the dorso-medial region, and on the septum and the nasomaxillary turbinates. The lesions in the group exposed to 300 ppm (127 mg/cu.m) were characterized by moderate atrophy of the olfactory epithelium accompanied by a thickened submucosa and moderate to marked basal-cell hyperplasia of the olfactory epithelium at 28 months only. Moderate to marked nest-like infolds of the respiratory epithelium was observed at 18, 24, and 28 months. At 100 ppm (43 mg/cu.m) slight basal cell hyperplasia of the olfactory epithelium was observed in female rats at 28 months and slight nest-like infolds of the respiratory epithelium in both males (18, 24, and 28 months) and females (24 and 28 months). This latter effect was the only respiratory change that was statistically significant in the animals exposed to 30 ppm (13 mg/cu.m) and was seen only in rats exposed for 28 months. No other non-neoplastic treatment-related effects were observed. This study identifies the LOAEL for extrathoracic respiratory tract effects at a concentration of 30 ppm [LOAEL(HEC) = 2.9 mg/cu.m].

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___I.B.3. UNCERTAINTY AND MODIFYING FACTORS (INHALATION RfC)

UF = 100. An uncertainty factor of 10 is used for protection of sensitive human subpopulations. A factor of 10 is used for interspecies extrapolation and to account for the use of a LOAEL because the effect is mild and occurs only at the 28-month exposure and not at the 24-month exposure.

MF = 1.

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___I.B.4. ADDITIONAL STUDIES / COMMENTS (INHALATION RfC)

NTP (1985) conducted a 2-year bioassay in F344 rats and B6C3F1 mice to determine the chronic toxic and carcinogenic effects of inhaled propylene oxide. Fifty animals/species/sex were exposed to 99.9% pure propylene oxide at concentrations of 0, 200, and 400 ppm (0, 475, and 950 mg/cu.m) for 6 hours/day, 5 days/week for 103 weeks (duration adjusted concentrations: 0, 85, and 170 mg/cu.m). Hematology, serum chemistry, urinalysis, and histopathology

were performed. Survival in the rats was unaffected by exposure to propylene oxide, and terminal body weights were slightly depressed in the high-dose male (8%) and female (9%) rats. In mice, survival tended to be adversely affected in all treated groups, but the decrease was statistically significant only for male and female mice in the 400 ppm group. Terminal body weights were 10% below control values for the high-dose female mice and 22% below control values for the high-dose male mice. The FEL for body weight changes is 400 ppm (FEL[HEC] = 170 mg/cu.m). The respiratory epithelium of the nasal turbinates was the primary tissue affected by propylene oxide exposure in both rats and mice. Rats exhibited exposure-related increases in suppurative inflammation of the nasal cavity (7/50, 19/50, and 33/50 in the control, 200, and 400 ppm males, respectively, and 3/50, 5/50, and 20/50 in the control, 200, and 400 ppm females, respectively) in addition to exposure-related increases in epithelial hyperplasia (0/50, 1/50, and 11/50 in males; 0/50, 0/48, and 5/48 in females in respective dose groups) and squamous metaplasia (1/50, 3/50, and 21/50 in males; 1/50, 2/48, and 11/48 in females in respective dose groups). Chronic inflammation of the nasal cavity was observed in 1/50, 13/50, and 38/50 of the male mice and in 0/50, 13/50, and 17/50 of the female mice exposed to 0, 200 ppm, and 400 ppm, respectively. Hyperplasia and metaplasia were also observed sporadically in mice exposed to 400 ppm propylene oxide. These lesions were most pronounced in the anterior portion of the nasal cavity and on the greater curvatures of the nasal maxillary turbinates. No consistent effect was observed in the tracheobronchiolar or pulmonary region of the respiratory tract, or in skeletal muscle, bronchial lymph nodes, or central nervous system. The LOAEL(ADJ) for extrathoracic respiratory effects for rats and mice was identified in this study as 200 ppm [LOAEL(HEC) = 16 mg/cu.m for rats (RGDR = 0.183 for female F-344 rat) and 16 mg/cu.m for mice (RGDR = 0.183 for female B6C3F1 mice)].

The chronic toxicity of inhaled propylene oxide was studied in male F344 rats by Lynch et al. (1984). Eighty rats/group were exposed to 0, 100, or 300 ppm propylene oxide (0, 238, and 713 mg/cu.m) for an average of 6.9 hours/day, 5 days/week for 104 weeks (duration adjusted concentrations, 0, 49, and 150 mg/cu.m). Body weight and survival were significantly reduced in the 300 ppm treatment group. Hemoglobin concentrations were significantly elevated above control values in both groups of propylene oxide-treated rats. Differential leukocyte counts were also elevated in the rats exposed to propylene oxide, but this effect is probably a result of the outbreaks of Mycoplasma pneumonia infection which occurred at 8, 16, and 20 months of the study. The authors state that the presence of Mycoplasma was confirmed by serology but do not indicate that the infection was confined to the exposed groups. None of the observed changes in absolute and relative organ weights were consistently associated with histopathological changes and were not considered to be treatment-related. Effects observed in exposed groups in this study included suppurative rhinitis and complex epithelial hyperplasia in the nasal cavity; pulmonary pneumonia and edema; tracheitis; bronchial lymph node hyperplasia; and multifocal myopathy of the skeletal muscle. Nasal suppurative rhinitis was increased significantly in both exposed groups. The nasal epithelial hyperplasia was statistically significant only in the 713 mg/cu.m group and was also observed in 2 animals in the 240 mg/cu.m group; it appears to be exposure related, although the effect of the mycoplasmosis is unknown. The respiratory effects cited above occurred with significantly greater frequency in both exposed groups. This study suggests a LOAEL of 100 ppm [LOAEL(HEC) = 13 mg/cu.m] for the extrathoracic respiratory effect and for the thoracic effect [LOAEL(HEC) = 163 mg/cu.m, RGDR = 3.33]. Skeletal muscle myopathy was increased in 25/78 rats exposed to 300 ppm compared with 7/77 controls, but was not indicated as statistically significant. This study indicates a LOAEL of 300 ppm [LOAEL(HEC) = 710 mg/cu.m] for skeletal myopathy and effects on body weight based on a systemic effect of a soluble gas.

NIOSH sponsored an investigation of the developmental toxicity of inhaled propylene oxide in Sprague-Dawley rats and rabbits (Hackett et al., 1982; Hardin et al., 1983). The study population consisted of 23-30 artificially inseminated rabbits per group and 32 to 45 sperm-positive rats per group. The number of litters examined ranged from 9 to 19 per group for the rabbits and 41 to 46 per group for the rats. All animals were exposed to 500 ppm propylene oxide (1188 mg/cu.m) for 7 hours/day. The protocol for the rats was: Group 1, control (filtered air); Group 2, propylene oxide on gestation days 7-16; Group 3, propylene oxide on gestation days 1-16; Group 4, propylene oxide for 3 weeks (5 days/week) prior to mating and daily on gestation days 1-16. The protocol for the rabbits was: Group 1, control (filtered air); Group 2, propylene oxide on gestation days 7-19; Group 3, propylene oxide on gestation days 1-19. The animals were necropsied and uterine contents were examined on gestation day 21 (rats) or 30 (rabbits). Maternal body weight gain and food consumption were decreased in all exposed rats. Reproductive capacity was impaired in rats exposed prior to breeding (Group 4). The number of corpora lutea, implantation sites, and live fetuses was reduced in rats exposed pregestationally to propylene oxide. Dams exposed during gestation (especially those exposed on gestation days 7-16) had more resorptions. Some degree of fetotoxicity was observed in all exposed groups of rats and included a significant reduction in fetal body weight and crown-rump length. The only evidence of fetal malformations seen in rats was an increase in wavy ribs and reduced ossification in rats exposed from gestation days 1-16. The incidence of rib dysmorphology and reduced ossification of fetal ribs and vertebrae were more frequent in exposed animals.

The only signs of maternal toxicity reported in the rabbits was a significant reduction in food consumption and a slight but significant decrease in maternal body weight gain in both groups exposed at some times during gestation (Hackett et al., 1982; Hardin et al., 1983). The only evidence of fetal toxicity observed in the rabbits was an increase in resorptions per litter, comparing only litters with resorptions from does exposed during gestation days 1-19 (Group 3), with no change in total resorptions. Reproductive measures, including number of corpora lutea, implantations, and live fetuses, were similar for all groups. Fetal size, sex ratio, and placental weights were unaffected by exposure in any group. Minor musculoskeletal anomalies (sternebral and limb anomalies) were significantly increased in fetal rabbits from Group 3. These studies suggest a LOAEL of 1188 mg/cu.m [LOAEL(HEC) = 1188 mg/cu.m] for reproductive and minor developmental effects, but it is not known whether these effects occurred as a result of maternal toxicity.

Harris et al. (1989) exposed groups of 25 mated female Fischer 344 rats to 0, 100, 300, or 500 ppm propylene oxide for (0, 238, 713, and 1188 mg/cu.m) 6 hours/day on gestation days 6-15. Reduced maternal body weight gain and food consumption was observed in the dams exposed to 500 ppm propylene oxide, similar to the NIOSH study findings. However, there was no exposure-related fetotoxicity (e.g., the number of viable fetuses or fetal body weight, postimplantation losses, total implantations, and corpora lutea) observed in this study, in contrast to the NIOSH study findings. The only fetal malformation observed was an increased frequency of seventh cervical rib in the 500 ppm group. The authors attribute the difference in results obtained between the two studies to a difference in strain susceptibility to the toxic effects of propylene oxide. This study identifies a NOAEL of 300 ppm [NOAEL(HEC) = 713 mg/cu.m] for minor developmental variations and maternal toxicity as measured by decreased weight gain.

Hayes et al. (1988) conducted a 2-generation reproductive toxicity study in F344 rats exposed to propylene oxide by inhalation. Thirty rats of each

sex (F0) were exposed to 0, 30, 100, or 300 ppm (0, 71, 238 , and 713 mg/cu.m) propylene oxide 6 hours/day, 5 days/week for 14 weeks prior to mating to produce the F1 litters. Thirty randomly selected F1 pups/sex/group were then exposed to the same concentrations of propylene oxide for 17 weeks after weaning and subsequently mated to produce the F2 generation. Data on body weight, litter size, live pups, litter weight was collected, and complete histopathology on F1 and F2 pups (10 pups/sex/exposure) in the control and 300 ppm groups. Body weight was significantly decreased in F0 (94% and 92% of control in females and males respectively) and F1 (89% of control in males and females) rats exposed to 713 mg/cu.m propylene oxide. No treatment-related effect was observed in any of the following reproductive parameters: fertility, litter size and neonatal growth, and survival. Furthermore, no effects attributable to propylene oxide exposure were observed at gross pathological examination in either the adults or weanlings or histopathological examination of the pups. This study identifies a NOAEL for reproductive effects of 300 ppm [NOAEL(HEC) = 713 mg/cu.m] and a NOAEL for changes in body weight of 100 ppm [NOAEL(HEC) = 238 mg/cu.m].

Functional and histopathological evidence of neurotoxicity in Wistar rats exposed to 1500 ppm propylene oxide (3563 mg/cu.m) for 6 hours/day, 5 days/week for 7 weeks (Ohnishi et al., 1988) (duration-adjusted concentration = 636 mg/cu.m). Awkward gait was apparent in exposed rats by the third to fourth week of exposure and all rats exhibited obvious ataxia by the seventh week. Histopathological examination revealed axonal degeneration of the hindleg nerve and fasciculus gracilis myelinated fibers, and myelinated fibers in the sacral spinal root. The LOAEL(HEC) for this study is 636 mg/cu.m.

Sprinz et al. (1982) exposed male cynomolgus monkeys for 2 years (2/group) to 0, 100, or 300 ppm propylene oxide (0, 237, and 712 mg/cu.m). Nerve conduction velocity was measured throughout the exposure and at the termination of exposure, sections of peripheral nerves, spinal cord, and brain (19 regions) were examined. No exposure-related changes were observed in the peripheral nerves or the spinal cord. Axonal dystrophy was observed in the medulla oblongata and in the most distal portions of the fasciculus gracilus in one control monkey and in all four exposed monkeys. The extent of the lesion was similar in all affected monkeys and was not dose-related.

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___I.B.5. CONFIDENCE IN THE INHALATION RfC

Study: Medium
Data Base: Medium
RfC: Medium

The study by Kuper et al. (1988) used a large number of animals, examined the critical effect with sensitive techniques and at multiple durations and exposure levels, and was of chronic duration, but did not identify a NOAEL, resulting in medium confidence. There are several corroborative chronic inhalation studies and inhalation developmental studies, but the inhalation 2-generation reproductive study is inadequate, resulting in medium confidence in the data base. Medium confidence in the RfC results.

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___I.B.6. EPA DOCUMENTATION AND REVIEW OF THE INHALATION RfC

Source Document -- This assessment is not presented in any existing U.S. EPA document.

Other EPA Documentation -- U.S. EPA, 1985, 1987

Agency RfD Work Group Review: 06/21/90, 09/20/90

Verification Date: 09/20/90

___I.B.7. EPA CONTACTS (INHALATION RfC)

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_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- Propylene oxide
CASRN -- 75-56-9
Last Revised -- 02/01/91

Section II provides information on three aspects of the carcinogenic risk assessment for the agent in question; the U.S. EPA classification, and quantitative estimates of risk from oral exposure and from inhalation exposure. The classification reflects a weight-of-evidence judgment of the likelihood that the agent is a human carcinogen. The quantitative risk estimates are presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per (mg/kg)/day. The unit risk is the quantitative estimate in terms of either risk per ug/L drinking water or risk per ug/cu.m air breathed. The third form in which risk is presented is a drinking water or air concentration providing cancer risks of 1 in 10,000, 1 in 100,000 or 1 in 1,000,000. Background Document 2 (Service Code 5) provides details on the rationale and methods used to derive the carcinogenicity values found in IRIS. Users are referred to Section I for information on long-term toxic effects other than carcinogenicity.

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__II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

___II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification-- B2; probable human carcinogen

Basis-- Based on inadequate human data and an increased incidence of benign and malignant tumors at the site of exposure in two species of animals, when exposed by subcutaneous injection, by inhalation, and by gavage. There was also evidence of mutagenicity in a variety of test systems. Propylene oxide is structurally similar to other chemicals that demonstrate carcinogenic activity in animals.

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___II.A.2. HUMAN CARCINOGENICITY DATA

Inadequate. Theiss et al. (1982) conducted a retrospective cohort study of 602 employees in eight German production plants, where there was exposure to alkylene oxides (propylene oxide and ethylene oxide) and other chemicals, including dichloropropane and epichlorohydrin. The mortality in each cancer category was not significantly higher than expected.

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___II.A.3 ANIMAL CARCINOGENICITY DATA

Sufficient. The animal data consist of oral, inhalation and subcutaneous studies in three strains of rats and two strains of mice. Propylene oxide caused tumors at or near the site of administration in rodents, causing forestomach tumors following ingestion (Dunkelberg, 1982) and nasal tumors after inhalation exposure (NTP, 1985).

Dunkelberg (1982) treated groups of 50 female Sprague-Dawley rats by gavage with 0, 15 or 60 mg/kg of 1,2-propylene oxide in salad oil twice weekly for 150 weeks (a total of 219 treatments) for total average doses of 0, 2714, or 10,798 mg/kg, as reported by the author. (Treatments were temporarily suspended due to a pneumonia outbreak.) All animals were treated with antibiotics when pneumonia interrupted treatment for 3 weeks. Controls consisted of both vehicle-treated and untreated groups. Survival of treated animals was comparable to controls with approximately 30% mortality at 104 weeks. Forestomach tumors, primarily squamous cell carcinomas, first developed in treated animals during week 79 in the high-dose group. The incidence of squamous cell carcinomas of the forestomach was 0/100 (combined untreated and vehicle controls), 2/50 (low-dose) and 19/50 (high-dose). No statistical analyses were conducted. The incidence of tumors at sites distant from the site of administration appeared to be similar in treated and untreated groups.

F344/N rats and B6C3F1 mice (50/sex/dose) were exposed by inhalation to 0, 200, or 400 ppm (0, 475, or 950 mg/cu.m) of propylene oxide for 6 hours/day, 5 days/week for 103 weeks (NTP, 1985; Renne et al., 1986). Survival of treated rats was comparable to that of controls. Dose-related increases in inflammation and metaplasia of the nasal cavities in both sexes of rats and a positive trend in papillary adenomas of the nasal turbinate epithelium (0/50, 0/50, 3/50) in female rats were reported. Incidence of thyroid gland C-cell adenoma or carcinomas (2/45, 2/35, 7/37) in female rats showed a positive trend that was statistically significantly elevated in the high-dose group relative to controls, when the incidences of benign and malignant tumors were combined (there were equal proportions of both tumor types). The incidence of endometrial stromal polyps and sarcomas combined (3/49, 12/50, 10/47) (the majority was polyps) was statistically significantly greater in rats at all doses compared to controls inhaling propylene oxide than in controls. In male rats, a significant positive trend for keratoacanthomas occurred (1/50, 1/50, 5/50). The NTP, however, considered only the nasal cavity tumors as being treatment-related because the other tumors were either a relatively common type (thyroid) or were of low incidence relative to that in historical controls.

Nineteen weeks after the initial start date, a technical error caused excessive chamber concentration, killing all low-dose mice. New groups of low-dose mice of each sex were started, but additional control groups were not included. Survival in mice showed dose-related decreases in both sexes with a statistically significant decrease in high-dose animals relative to controls. At termination, 58% of males and 20% of females in the high-dose group survived as compared with 84 and 76% of the respective controls. Terminal body weights of high-dose animals were decreased by 10-21%,

indicating a MTD had been achieved. One squamous cell carcinoma and one papilloma in the nasal cavity of two high-dose males, and nasal cavity adenocarcinomas of two high-dose females were reported. The incidences of these lesions (combined) were 0/50 and 0/1615 in males, and 0/50 and 0/1668 in females for concurrent and historical control mice of this strain in the NTP program, respectively. Hemangiomas (0/50, 0/50, 5/50) and hemangiosarcomas (0/50, 0/50, 5/50) of the nasal cavity were statistically significantly increased both individually and when combined in male mice receiving 400 ppm propylene oxide when compared with concurrent controls. In females, the incidence of combined hemangiomas or hemangiosarcomas of the nasal cavity (0/50, 0/50, 5/50) was also statistically significantly elevated at the high dose. Statistical analysis of these tumors included adjustment for intercurrent mortality. When compared by life table tests, mammary gland adenocarcinomas (all types) showed a significant dose-related trend and significant elevation in high-dose females relative to controls (0/50, 3/50, 3/50).

One hundred Cpb:WU Wistar rats/sex/group were exposed by inhalation to 0, 30, 100, or 300 ppm (0, 71, 238, or 713 mg/cu.m) propylene oxide for 6 hours/day, 5 days/week for 123 to 124 weeks (Reuzel and Kuper, 1983; Kuper et al., 1988). Interim sacrifices of 10 rats/sex/group were conducted at 12, 18, and 24 months. Survival was significantly decreased in high-dose animals relative to controls with only 21 to 47% of treated males and females surviving after week 115, as compared with 54 to 71% of controls. By week 119, survival in females at the mid-dose level, 100 ppm, was also significantly decreased (survival was 39%). While there were no tumors in the nasal cavity, a statistically significant increase in nonneoplastic alterations (degenerative changes and hyperplasia) of the olfactory and respiratory epithelium was observed in each sex in each exposure group. A statistically significant increase in mammary gland fibroadenomas (32/69, 30/71, 39/69, 47/70) and adenocarcinomas (3/69, 6/71, 5/69, 8/70), which appeared earlier in life, were found in the high-dose females when compared with controls. The authors suggested malignant mammary tumor development in the high-dose females may not be related to propylene oxide exposure, since the historical control incidence of malignant mammary tumors in this laboratory is in the range of 0 to 15% (0/30 to 15/99). Although not statistically significantly elevated, squamous-cell carcinomas of the nose, larynx/pharynx and trachea, and adenocarcinomas of the larynx/pharynx and lungs were reported in five high-dose males; none of these tumor types were reported in control males.

Lynch et al. (1984a) exposed male F344 rats (80/group) to 0, 100, or 300 ppm propylene oxide 7 hours/day, 5 days/week for 104 weeks. A statistically significant increase in mortality was observed in each treated group relative to controls. At 104 weeks, survival was approximately 50, 45, and 35% for the control, low-dose and high-dose groups, respectively. An outbreak of mycoplasmosis at approximately 68 weeks contributed to the decreased survival of all animals. A statistically significant increase in hyperplasia of the nasal epithelium was reported in high-dose rats. The incidence of adrenal pheochromocytomas (8/78, 25/78, 22/80), although without increasing trend, was statistically significantly elevated in each group of the propylene oxide-exposed rats (not explicit when a trend test was conducted).

Dunkelberg (1981) treated groups of 100 female NMRI mice weekly by subcutaneous injection with 0.1, 0.3, 1.0, or 2.5 mg propylene oxide in tricaprylin once a week for 95 weeks for average total doses of 6.8, 21.7, 72.8, 165.4 mg/mouse. Controls consisted of groups of 100 untreated or vehicle-treated mice. A dose-related increase (approximately 2 to 16%) in injection site tumors (mostly fibrosarcomas) was reported with the first tumor appearing in the high-dose group at 39 weeks, while no tumors were noted among

vehicle or untreated controls. Tumor incidence at other sites was similar in control and treated groups.

Propylene oxide in either arachis oil or water was injected subcutaneously in groups of 12 rats (sex and strain not specified) over 325 days for a total dose of 1500 mg/kg (Walpole, 1958). Injection site sarcomas developed in 8/12 and 3/12 rats receiving propylene oxide in the oil and water vehicles, respectively. No controls appear to have been run and no other experimental details were available.

<<< Propylene oxide >>>

___II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Like other epoxides, propylene oxide is DNA-reactive. Propylene oxide has been found to be mutagenic in a variety of test systems. Reverse mutations were produced in Salmonella typhimurium both with and without mammalian hepatic homogenates as a metabolic activating system (Wade et al., 1978; Bootman et al., 1979; McMahon et al., 1979; Hemminki and Falck, 1979; Pfeiffer and Dunkelberg, 1980; Yamaguchi, 1982; Yamaguchi and Nakagawa, 1983; Djuric et al. 1986) and Escherichia coli (Bootman et al., 1979; McMahon et al., 1979; Hemminki et al., 1980), and without mammalian hepatic homogenates as a metabolic activating system in Klebsiella pneumoniae (Voogd et al., 1981). Forward mutations were produced in Schizosaccharomyces pombe (Migliore et al., 1982) and chinese hamster ovary cells (Zamora et al., 1983). Propylene oxide induced sex-linked recessive lethal mutations in Drosophila melanogaster (Hardin et al., 1983). Chromosome aberrations (Dean and Hodson-Walker, 1979) and DNA strand breaks (Sina et al., 1983) have been reported in rat hepatocytes, but no significant increases in sister-chromatid exchange (SCE) or chromosomal aberrations in lymphocytes from Cynomolgus monkeys were reported by Lynch et al. (1984b). Bootman et al. (1979) reported increased chromosomal aberrations and Tucker et al. (1986) found increased SCE frequency in human lymphocytes. DNA synthesis and repair was reduced in lymphocytes of workers occupationally exposed to less than 12 ppm propylene oxide for 2 to 20 years (Pero et al., 1982) and lymphocyte chromosome aberrations were increased in workers exposed to alkylene oxides (ethylene oxide and propylene oxide) for >20 years compared with a presumably unexposed group of workers. No baseline values were available for the exposed workers (Theiss et al., 1981).

Propylene oxide is structurally-related to epichlorohydrin and ethylene oxide, which have induced carcinogenic responses in animals. Limited human evidence for cancer risk also exists for ethylene oxide.

---------<<< Propylene oxide >>>---------

__II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSURE

___II.B.1. SUMMARY OF RISK ESTIMATES

Oral Slope Factor-- 2.4 E-1 per (mg/kg)/day

Drinking Water Unit Risk-- 6.8E-6 per (ug/L)

Extrapolation Method -- Linearized multistage procedure, extra risk

Drinking Water Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	1E+1 ug/L
E-5 (1 in 100,000)	1E+0 ug/L
E-6 (1 in 1,000,000)	1E-1 ug/L

<<< Propylene oxide >>>

___II.B.2. DOSE-RESPONSE DATA (CARCINOGENICITY, ORAL EXPOSURE)

Tumor Type -- forestomach, squamous cell carcinoma
Test Animals -- rat/Sprague-Dawley, female
Route -- gavage, salad oil
Reference -- Dunkelberg, 1982

Dose: Administered (mg/kg)	Dose: Transformed Animal Dose (mg/kg/day)	Human Equivalent (mg/kg/day)	Tumor Incidence
0	0	0	0/100
15	2.58	0.44	2/50
60	10.28	1.76	19/50

<<< Propylene oxide >>>

___II.B.3. ADDITIONAL COMMENTS (CARCINOGENICITY, ORAL EXPOSURE)

Human equivalent doses were determined using a rat body weight of 0.35 kg, a human body weight of 70 kg, 1029 days as the length of the exposure, and 1050 days as the length of the experiment and lifespan of the animal.

The unit risk should not be used if the water concentration exceeds 1E+3 ug/L, since above this concentration the unit risk may not be appropriate.

<<< Propylene oxide >>>

___II.B.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, ORAL EXPOSURE)

The study was conducted for a long period of time (150 weeks) with a sufficient number of animals surviving for analysis of late-developing tumors. Survival of treated animals was comparable with that of controls at 104 weeks; both had approximately 30% mortality. Exposure was by a relevant route and a comprehensive necropsy was performed.

---------<<< Propylene oxide >>>---------

__II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION EXPOSURE

___II.C.1. SUMMARY OF RISK ESTIMATES

Inhalation Unit Risk-- 3.7E-6 per (ug/cu.m.)

Extrapolation Method-- Linearized multistage procedure, extra risk

Air Concentrations at Specified Risk Levels

Risk Level	Concentration
E-4 (1 in 10,000)	3E+1 ug/cu.m
E-5 (1 in 100,000)	3E+0 ug/cu.m
E-6 (1 in 1,000,000)	3E-1 ug/cu.m

<<< Propylene oxide >>>

___II.C.2. DOSE-RESPONSE DATA FOR CARCINOGENICITY, INHALATION EXPOSURE

Tumor Type -- nasal cavity hemangioma or hemangiosarcoma
Test Animals -- mouse/B6C3F1, male
Route -- Inhalation
Reference -- NTP, 1985; Renne et al., 1986

---- Dose ----- Administered (ppm)	Transformed Animal Dose (mg/kg)/day	Human Equivalent (mg/kg)/day	Tumor Incidence
0	0	0	0/50
200	55	4.15	0/50
400	110	8.29	10/50

<<< Propylene oxide >>>

___II.C.3. ADDITIONAL COMMENTS (CARCINOGENICITY, INHALATION EXPOSURE)

Transformed animal doses were calculated assuming 50% pulmonary absorption exposure duration and length of experiment of 103 weeks. The 50% absorption assumption is consistent with the absorption efficiency observed for epichlorohydrin in the respiratory tract of rats (Stott and Mckenna, 1984). The transformed animal dose level was used in the calculation of an animal slope factor of 9.8E-4 per (mg/kg)/day. The human slope factor, 1.3E-2 per (mg/kg)/day was determined using an animal body weight of 0.03 kg, a human body weight of 70 kg and animal lifespan of 103 weeks.

The above unit risk should not be used if the air concentration exceeds 3E+3 ug/cu.m., as above this concentration the unit risk may not be appropriat

<<< Propylene oxide >>>

___II.C.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, INHALATION EXPOSURE)

Adequate numbers of animals of both sexes of mice were treated for a lifetime. Propylene oxide exposure was by a relevant route and a complete histopathological examination was performed.

---------<<< Propylene oxide >>>---------

__II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASSESSMENT)

___II.D.1. EPA DOCUMENTATION

U.S. EPA. 1985. Health and Environmental Effects Profile for Propylene Oxide. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste, Washington, DC.

U.S. EPA. 1987. Summary Review of the Health Effects Associated with Propylene Oxide. Office of Health and Environmental Assessment, Washington, DC. EPA/600/8-86/007F.

<<< Propylene oxide >>>

___II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

The 1985 Health and Environmental Effects Profile and 1987 Health Issue Assessment for Propylene Oxide have received Agency review.

Agency Work Group Review: 05/30/89, 04/05/90

Verification Date: 04/05/90

___II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Fred Hauchman / OAQPS -- (919)541-5339 / FTS 629-5339

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_III. HEALTH HAZARD ASSESSMENTS FOR VARIED EXPOSURE DURATIONS

__III.A. DRINKING WATER HEALTH ADVISORIES

Substance Name -- Propylene oxide
CASRN -- 75-56-9

Not available at this time.

__III.B. OTHER ASSESSMENTS

Substance Name -- Propylene oxide
CASRN -- 75-56-9

Content to be determined.

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_IV. U.S. EPA REGULATORY ACTIONS

Substance Name -- Propylene oxide
CASRN -- 75-56-9

Not available at this time.

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_V. SUPPLEMENTARY DATA

Substance Name -- Propylene oxide
CASRN -- 75-56-9

Not available at this time.

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_VI. BIBLIOGRAPHY

Substance Name -- Propylene oxide
CASRN -- 75-56-9
Last Revised -- 08/01/91

__VI.A. ORAL RfD REFERENCES

None

---------<<< Propylene oxide >>>---------

__VI.B. INHALATION RfD REFERENCES

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VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

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---------<<< Propylene oxide >>>---------

__VI.D. DRINKING WATER HA REFERENCES

None

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SYNONYMS

Substance Name -- Propylene oxide
CASRN -- 75-56-9
Last Revised -- 10/01/90

75-56-9
AD 6 (SUSPENDING AGENT)
CASWELL NO. 713A
EPA PESTICIDE CHEMICAL CODE 042501
EPOXYPROPANE
1,2-EPOXYPROPANE
ETHYLENE OXIDE, METHYL-
HSDB 173
METHYL ETHYLENE OXIDE
METHYL OXIRANE
METHYLOXIRANE
NCI-C50099
OXIDO DE PROPILENO [SPANISH]
OXIRANE, METHYL-
OXYDE DE PROPYLENE [FRENCH]
PROPANE, EPOXY-
PROPANE, 1,2-EPOXY-
PROPENE OXIDE
PROPYLENE EPOXIDE
PROPYLENE OXIDE
1,2-PROPYLENE OXIDE
UN 1280