Aramite; CASRN 140-57-8

Human health assessment information on a chemical substance is included in the IRIS database only after a comprehensive review of toxicity data, as outlined in the IRIS assessment development process. Sections I (Health Hazard Assessments for Noncarcinogenic Effects) and II (Carcinogenicity Assessment for Lifetime Exposure) present the conclusions that were reached during the assessment development process. Supporting information and explanations of the methods used to derive the values given in IRIS are provided in the guidance documents located on the IRIS website.

STATUS OF DATA FOR Aramite

File First On-Line 06/01/1991

Category (section)	Assessment Available?	Last Revised
Oral RfD (I.A.)	not evaluated	
Inhalation RfC (I.B.)	not evaluated	
Carcinogenicity Assessment (II.)	yes	06/01/1991

I. Chronic Health Hazard Assessments for Noncarcinogenic Effects

I.A. Reference Dose for Chronic Oral Exposure (RfD)

Substance Name — Aramite
CASRN — 140-57-8

Not available at this time.

I.B. Reference Concentration for Chronic Inhalation Exposure (RfC)

Substance Name — Aramite
CASRN — 140-57-8

Not available at this time.

II. Carcinogenicity Assessment for Lifetime Exposure

Substance Name — Aramite
CASRN — 140-57-8
Last Revised — 06/01/1991

Section II provides information on three aspects of the carcinogenic assessment for the substance in question; the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates of risk from oral exposure and from inhalation exposure. 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. The rationale and methods used to develop the carcinogenicity information in IRIS are described in The Risk Assessment Guidelines of 1986 (EPA/600/8-87/045) and in the IRIS Background Document. IRIS summaries developed since the publication of EPA's more recent Proposed Guidelines for Carcinogen Risk Assessment also utilize those Guidelines where indicated (Federal Register 61(79):17960-18011, April 23, 1996). Users are referred to Section I of this IRIS file for information on long-term toxic effects other than carcinogenicity.

II.A. Evidence for Human Carcinogenicity

II.A.1. Weight-of-Evidence Characterization

Classification — B2; probable human carcinogen

Basis — Based on no human data and sufficient data from animal bioassays including increased incidence of liver tumors and/or neoplastic nodules in three strains of male and female rats and males of one strain of mice, and extrahepatic biliary system tumors in dogs following chronic oral exposure.

II.A.2. Human Carcinogenicity Data

None.

II.A.3. Animal Carcinogenicity Data

Sufficient. Popper et al. (1960) (see also Oser and Oser, 1962) fed 50 FDRL rats/sex/group 100, 200 or 400 ppm aramite in the diet for 104 weeks. Controls consisted of 100 rats/sex fed a basal diet containing no aramite. Weight gain reportedly was similar in all groups. Survival in all groups was 95% or greater in the first year of the study. At the end of the study, survival in the males was 59, 50, 46 and 46% in the control, low-, mid- and high-dose groups, respectively. In females survival was 61, 64, 40 and 34%, in the control, low-, mid-, and high-dose groups, respectively. Tumor incidence data were not reported by sex; however, no sex differences were noted in the pathology. There was a statistically significant dose-related increase in the incidence of hyperplastic liver nodules: 2/193, 2/93, 3/100 and 20/90 in rats (male and female data combined) in the control, low-, mid- and high-dose groups, respectively. The hyperplastic nodules described by Popper et al. (1960) would now be classified as neoplastic liver nodules (Baggs, 1990; Chiu and Singh, 1990). Also, in the high-dose group two liver carcinomas and five bile duct adenomas were found; these tumor types were not observed in any other groups. Rats with carcinomas also had neoplastic nodules (hence were counted in the incidence data above), but it is unclear whether the rats with the bile duct adenomas also had neoplastic liver nodules.

Popper et al. (1960) (see also Oser and Oser, 1962) also fed groups of 50 CFN rats/sex and 50 Sprague-Dawley rats/sex 100, 200 or 400 ppm aramite in the diet for 104 weeks. Controls consisted of 100 rats/sex/strain fed a diet containing no aramite. Tumor incidence data were not reported by sex. CFN rats showed a dose-related increased incidence of neoplastic nodules; 5/180, 3/93, 10/90 and 22/96 in the 0, 100, 200 or 400 ppm groups, respectively. No liver carcinomas were observed in CFN rats, but the incidences of bile duct adenomas were 0/180, 2/93, 1/200 and 2/96 in the control, 100, 200 and 400 ppm groups, respectively. It is unclear whether these rats also had neoplastic liver nodules. Only Sprague-Dawley rats dying after 1 year were examined. High mortality (about 60%) (unrelated to treatment) in the ninth and tenth months due to respiratory infections in the Sprague-Dawley rats precluded evaluation for late-developing tumors. Respiratory infections also caused many deaths in CFN rats (about 20%) (unrelated to treatment) after 6 months.

Oser and Oser (1960) fed groups of 20-21 FDRL rats (approximately 10/sex/group) diets containing 0, 500, 1580 or 5000 ppm aramite for 24-96 weeks. All rats fed the 5000 ppm diet died by 96 weeks of treatment, and survival was reduced in the other treated groups, compared with controls. Tumor incidence data were not reported by sex. The authors reported a significantly increased incidence of liver tumors: 0/20, 0/20, 2/21 and 6/20 in rats fed 0, 500, 1580 and 5000 ppm, respectively. Tumors in rats fed the 5000 ppm diet were described as hepatomas or cholangiomas; tumors in rats fed the 1580 ppm diet were described as malignancies. One rat fed the 500 ppm diet had neoplastic liver nodules. Neoplastic nodules

were not observed in the controls; the incidence of nodules in rats fed higher doses of aramite was not reported.

Male Wistar rats were fed diets containing aramite at 0 (20 rats) or 5000 ppm (33 rats) for 56 weeks (Truhaut et al., 1975, 1978; Blanc et al., 1978). Neither survival nor liver tumor incidence data were reported for the controls. At 56 weeks, 19/33 treated rats were alive and all (19/19) had liver tumors.

Radomski et al. (1965) fed 50 ppm aramite alone in the diet or 50 ppm aramite mixed with three other pesticides in the diet to 50 Osborne-Mendel rats/sex/group for 2 years. In a second experiment, 30 Osborne-Mendel rats/sex/group were fed 80 ppm aramite alone in diet or 80 ppm aramite mixed with three other pesticides in the diet for 2 years. Deichmann et al. (1967) fed 200 ppm aramite either alone or mixed with three other pesticides in the diet to 30 Osborne-Mendel rats/sex/group for 24-27 months. Although actual data were not provided, both studies stated that survival and weight gain were not affected by the dietary addition of aramite either alone or in combination [except for decreased weight gain in the group that was fed the mixture of four pesticides in the Deichmann et al. (1967) report]. In both reports, tumors were detected by gross examination at autopsy; all tumors and several body organs, including the liver, were examined histologically. There were no increases in the tumor incidence in rats fed aramite alone or in combination with other pesticides. Since the MTD was not reached in either study, both studies are considered inadequate.

Oser and Oser (1962) fed groups of 50 C3H mice/sex and 50 C57BL mice/sex 100, 200 or 400 ppm aramite in the diet for 2 years. Controls consisted of 100 mice/sex/strain fed a diet containing no aramite. There was no evidence of a neoplasia reported in either strain of mice.

In an extended regimen, B6C3F1 mice and B6AKF1 mice (16 mice/sex/strain) received aramite in 0.5% gelatin by gavage at 464 mg/kg/day from 7 days after birth until weaning at 4 weeks (Innes et al., 1969). After weaning, aramite was administered in the diet at 1112 ppm for approximately 80 weeks. A significant increase (6/16) in hepatomas was observed in male (C57BL/6xC3H/Anf)F1 mice compared with the incidence in controls (8/73). Tumor incidences in the female mice of this strain and in both sexes of the B6AKF1 strain were not increased by comparison to controls.

Oser and Oser (1960) fed mongrel dogs (3/group, sex not stated) a basal diet containing 0, 500 or 1580 ppm aramite. The dogs were sacrificed after 1 year of study. Autopsies showed degeneration of the liver; however, no tumors were reported. This study is inadequate due to small number of animals and the short duration of the experiment.

Sternberg et al. (1960) (see also Oser and Oser, 1962) identified tumors in the extrahepatic biliary tract of dogs exposed to aramite in the diet. A total of 40 mongrel dogs (17 male and 23 female) were fed diets containing aramite at 0, 500 or 828-1429 ppm for 811-1220 days (low-dose group) and for 462-1206 days (high-dose group). Seven of the 12 low-dose dogs and 12/16 high-dose dogs appeared moribund or died during treatment and were necropsied and examined for tumors. The control dogs and the remaining low-dose dogs appeared healthy and were not autopsied. Extrahepatic biliary system adenocarcinomas were found in 7/7 low-dose dogs and in 7/12 high-dose dogs subjected to necropsy (extrahepatic biliary system adenocarcinomas were observed in all of the 7 high-dose dogs surviving more than 715 days on study). Neoplastic nodules in the liver parenchyma (3/7 and 3/12 in the low- dose and high-dose dogs, respectively), and hyperplasia and adenocarcinomas of liver bile ducts (6/7 and 7/12 in the low-dose and high-dose dogs, respectively) were also observed in both treated groups. Five of the 12 high- dose dogs died early in the experiment (before 715 days) with no signs of cancer. No statistical analyses were reported.

Hodge et al. (1966) administered a single subcutaneous injection of 10 mg aramite in the vehicle trioctanonin to 50 C3H/Anf mice/sex. Vehicle controls, 50 C3H/Anf mice/sex, were injected with trioctanonin only. The body weights of aramite-injected and vehicle-control groups did not differ significantly. Among the male groups the mean survival times were not significantly different. The mean survival times of aramite-injected females (401 days) were greater than their corresponding vehicle-controls (337 days). (The statistical significance of this reduction in survival was not reported.) Many of these deaths were due to pneumonitis. In aramite-injected mice there was no evidence of injection-site tumors during gross examination or of neoplasia during histological examination.

In a dermal application study, Hodge et al. (1966) applied either 0.1 mg of aramite or 10 mg of aramite in acetone weekly to the shaved skins of 50 C3H/Anf mice/sex. Two acetone control groups/sex (one corresponding to each dose) were also utilized. The mice were housed 12-13/cage. The mean survival time of the low-dose males (426 days) was equivalent to those of their corresponding controls (430 days). The mean survival time of all other groups differed significantly from corresponding control groups; the mean survival time for high-dose males was 452 days vs. 386 in the appropriate controls, for low-dose females it was 328 days vs. 393 in the appropriate controls, and for high-dose females it was 441 days vs. 313 in the appropriate controls. Many of these deaths were due to pneumonitis and were not the result of chemical-related toxicity. No gross evidence of tumor formation in the skins of control or treated mice was detected.

II.A.4. Supporting Data for Carcinogenicity

Aramite was negative for mutagenicity expressed as a dominant lethal effect when administered as a single intraperitoneal injection of 200 or 500 mg/kg dose into 7 and 9 male ICR Ha Swiss mice, respectively (Epstein et al., 1972).

II.B. Quantitative Estimate of Carcinogenic Risk from Oral Exposure

II.B.1. Summary of Risk Estimates

Oral Slope Factor — 2.5E-2 per (mg/kg)/day

Drinking Water Unit Risk — 7.1E-7 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+2 ug/L
E-5 (1 in 100,000)	1E+1 ug/L
E-6 (1 in 1,000,000)	1E+0 ug/L

II.B.2. Dose-Response Data (Carcinogenicity, Oral Exposure)

Tumor Type — neoplastic liver nodules and carcinomas
Test Animals — rat/FDRL, male and female
Route — diet
Reference — Popper et al., 1960; Oser and Oser, 1962

---------- Dose ----------			
Administered (ppm)	**Transformed Animal (mg/kg)/day**	**Human Equivalent (mg/kg)/day**	**Tumor Incidence**
0	0	0	2/193
100	5	0.78	2/93
200	10	1.57	3/100
400	20	3.14	20/90

II.B.3. Additional Comments (Carcinogenicity, Oral Exposure)

This study yielded the most appropriate quantitative estimate of cancer risk for aramite. Carcinoma and neoplastic hyperplastic liver nodule incidences were combined; however, carcinomas were observed only in two rats in the high-dose group.

The transformed animal dose was calculated by multiplying the administered dose (in ppm) by the food factor, assumed to be 0.05 for rats. The human equivalent dose was calculated by multiplying the transformed animal dose by the cube route of the ratio of the body weight of the rats (estimated at 0.270 kg) to the assumed body weight of humans (70 kg).

The unit risk should not be used if the water concentration exceeds 1E+4 ug/L, since above this concentration the slope factor may differ from that stated.

II.B.4. Discussion of Confidence (Carcinogenicity, Oral Exposure)

The key study was conducted with a sufficient number of animals of both sexes using a relevant route of exposure and control plus three doses, the highest of which was only slightly below the maximum tolerated dose, for the lifetime of the animals. The data demonstrated a dose relationship for tumor incidence. Data from dog studies indicate that this species may be more sensitive to the carcinogenic effects of aramite, but poor survival precluded the use of dog data for quantitation of risk.

II.C. Quantitative Estimate of Carcinogenic Risk from Inhalation Exposure

II.C.1. Summary of Risk Estimates

Inhalation Unit Risk — 7.1E-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)	1E+1 ug/cu.m
E-5 (1 in 100,000)	1E+0 ug/cu.m
E-6 (1 in 1,000,000)	1E-1 ug/cu.m

II.C.2. Dose-Response Data for Carcinogenicity, Inhalation Exposure

The inhalation estimates are derived from the oral exposure data presented in Section II.B.2.

II.C.3. Additional Comments (Carcinogenicity, Inhalation Exposure)

The inhalation unit risk and risk-specific concentrations in air were estimated from the oral data (see Section II) because inhalation data were not located. The observation of tumors at sites (the liver and extrahepatic biliary tract) distant from the digestive tract in oral studies in rats, mice and dogs supports the unit risk.

The unit risk should not be used if the air concentration exceeds 1E+4 ug/cu.m, above this concentration the unit risk may differ from that stated.

II.C.4. Discussion of Confidence (Carcinogenicity, Inhalation Exposure)

Data from inhalation exposures were not located; pharmacokinetic data are insufficient to predict whether the fates of inhaled and ingested aramite are similar.

II.D. EPA Documentation, Review, and Contacts (Carcinogenicity Assessment)

II.D.1. EPA Documentation

Source Document — U.S. EPA, 1989

The 1989 Health and Environmental Effects Document on Aramite has received external peer review and Agency Review.

II.D.2. EPA Review (Carcinogenicity Assessment)

Agency Work Group Review — 12/06/1990, 01/10/1991

Verification Date — 01/10/1991

Screening-Level Literature Review Findings — A screening-level review conducted by an EPA contractor of the more recent toxicology literature pertinent to the cancer assessment for aramite conducted in August 2003 did not identify any critical new studies. IRIS users who know of important new studies may provide that information to the IRIS Hotline at hotline.iris@epa.gov or 202-566-1676.

II.D.3. EPA Contacts (Carcinogenicity Assessment)

Please contact the IRIS Hotline for all questions concerning this assessment or IRIS, in general, at (202)566-1676 (phone), (202)566-1749 (FAX) or hotline.iris@epa.gov (internet address).

III. [reserved]
IV. [reserved]
V. [reserved]

VI. Bibliography

Substance Name — Aramite
CASRN — 140-57-8

VI.A. Oral RfD References

None

VI.B. Inhalation RfC References

None

VI.C. Carcinogenicity Assessment References

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U.S. EPA. 1989. Health and Environmental Effects Document for Aramite. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste, Washington, DC.

VII. Revision History

Substance Name — Aramite
CASRN — 140-57-8

Date	Section	Description
06/01/1991	II.	Carcinogenicity assessment on-line
10/28/2003	II.D.2.	Screening-Level Literature Review Findings message has been added.

VIII. Synonyms

Substance Name — Aramite
CASRN — 140-57-8
Last Revised — 06/01/1991

- 140-57-8
- SULFUROUS ACID, 2-(p-t-BUTYLPHENOXY)-1-METHYLETHYL-2-CHLOROETHYL ESTER
- Sulfurous acid, 2-(p-tert-butylphenoxy)-1-methylethyl 2-chloroethyl ester
- Sulfurous acid, 2-chloroethyl 2-(4-(1,1-dimethylethyl)phenoxy)-1-methylethyl ester
- 2-p-tert-butylphenoxyisopropyl 2-chloroethyl sulfite
- Acaracide
- AI3-16519
- Aracide
- Aramit
- Aramite
- Aramite-15W
- ARAMITEARARAMITE-15W
- Aratron
- beta-CHLOROETHYL-beta-(p-t-BUTYLPHENOXY)-alpha-METHYLETHYL SULPHITE
- beta-CHLOROETHYL-beta'-(p-t-BUTYLPHENOXY)-alpha'-METHYLETHYL SULFITE

- Butylphenoxyisopropyl chloroethyl sulfite
- Caswell No. 131
- CES
- Compound 88R
- ENT 16,519
- EPA Pesticide Chemical Code 062501
- ETHANOL, 2-CHLORO-, ESTER WITH 2-(p-tert-BUTYLPHENOXY)-1-METHYLETHYL SULFITE
- ETHANOL, 2-CHLORO-, 2-(p-t-BUTYLPHENOXY)-1-METHYLETHYL
- SULFITE
- Niagaramite
- NSC 404155
- Ortho-Mite
- SULFUROUS ACID, 2-(P-T-BUTYLPHENOXY)-1-METHYLETHYL-2-CHLOROETHYL ESTER
- SULFUROUS ACID, 2-(P-TERT-BUTYLPHENOXY)-1-METHYLETHYL 2-CHLOROETHYL ESTER
- SULFUROUS ACID, 2-CHLOROETHYL 2-(4-(1,1-DIMETHYLETHYL)PHENOXY)-1-METHYLETHYL ESTER
- SULFUROUS ACID, 2-CHLOROETHYL-, 2-(4-(1,1-DIMETHYLETHYL)PHENOXY)-1-METHYLETHYL ESTER
- 2-(p-butylphenoxy)-1-methylethyl 2-chloroethyl sulfite
- 2-(p-Butylphenoxy)isopropyl 2-chloroethyl sulfite
- 2-(p-t-BUTYLPHENOXY)-1-METHYLETHYL SULPHITE of 2-CHLOROETHANOL
- 2-(p-t-BUTYLPHENOXY)-1-METHYLETHYL 2-CHLOROETHYL ESTER of SULPHUROUS ACID
- 2-(p-t-BUTYLPHENOXY)-1-METHYLETHYL 2'-CHLOROETHYL SULPHITE
- 2-(p-t-BUTYLPHENOXY)-1-METHYLETHYL-2-CHLOROETHYL SULFITE
- 2-(p-t-butylphenoxy)isopropyl 2'-chloroethyl sulfite
- 2-(p-t-butylphenoxy)isopropyl 2'-chloroethyl sulphite
- 2-(p-terc.BUTYLFENOXY)ISOPROPYL-2'-CHLORETHYLESTER KYSELINY SIRICITE [Czech]
- 2-(p-tert-Butylphenoxy)-1-methylethyl sulfite of 2-chloroethanol
- 2-(p-tert-Butylphenoxy)-1-methylethyl 2-chloroethylsulfite
- 2-(p-tert-Butylphenoxy)-1-methylethyl 2'-chloroethyl sulfite
- 2-(p-tert-Butylphenoxy)-1-methylethyl-2-choroethyl sulfite
- 2-(p-tert-Butylphenoxy)isopropyl 2-chloroethyl sulfite
- 2-(4-t-BUTYLPHENOXY)ISOPROPYL-2-CHLOROETHYL SULFITE
- 2-(4-t-butylphenoxy)isopropyl-2-chloroethyl sulphite
- 2-(4-tert-Butylphenoxy)isopropyl 2-chloroethyl sulfite
- 2-(4-tert-Butylphenoxy)isopropyl-2-chloroethyl sulfite
- 2-Chloroethyl sulfite of 1-(p-t-butylphenoxy)-2-propanol
- 2-CHLOROETHYL SULPHITE of 1-(p-t-BUTYLPHENOXY)-2-PROPANOL
- 2-CHLOROETHYL 1-METHYL-2-(p-t-BUTYLPHENOXY)ETHYL SULPHATE
- 2-Chloroethyl 1-methyl-2-(p-tert-butylphenoxy)ethyl sulfite

- 2-PROPANOL, 1-(p-t-BUTYLPHENOXY)-, 2-CHLOROETHYL SULFITE
- 2-Propanol, 1-(p-tert-butylphenoxy)-, 2-chloroethyl sulfite
- 88-R
- 88R