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16. ABSTRACT

This report summarizes and evaluates information relevant to a preliminary interim assessment of adverse health effects associated with specific chemicals or compounds. The Office of Emergency and Remedial Response (Superfund) uses these documents in preparing cost-benefit analyses under Executive Order 12991 for decision-making under CERCLA. All estimates of acceptable intakes and carcinogenic potency presented in this document should be considered as preliminary and reflect limited resources allocated to this project. The intent in these assessments is to suggest acceptable exposure levels whenever sufficient data are available. The interim values presented reflect the relative degree of hazard associated with exposure or risk to the chemical(s) addressed. Whenever possible, two categories of values have been estimated for systemic toxicants (toxicants for which cancer is not the endpoint of concern). The first, RfD_S or subchronic reference dose, is an estimate of an exposure level that would not be expected to cause adverse effects when exposure occurs during a limited time interval. The RfD is an estimate of an exposure level that would not be expected to cause adverse effects when exposure occurs for a significant portion of the lifespan. For compounds for which there is sufficient evidence of carcinogenicity, q_1^*s have been computed, if appropriate, based on oral and inhalation data if available.

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HEALTH EFFECTS ASSESSMENT FOR ACENAPHTHENE

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PREFACE

This report summarizes and evaluates information relevant to a preliminary interim assessment of adverse health effects associated with acenaphthene. Pertinent toxicologic and environmental data were located through on-line literature searches of the Chemical Abstracts, TOXLINE, CANCERLINE and the CHEMFATE/DATALOG data bases. The basic literature searched supporting this document is current up to May, 1986. Secondary sources of information have also been relied upon in the preparation of this report and represent large-scale health assessment efforts that entail extensive peer and Agency review. The following Office of Health and Environmental Assessment (OHEA) sources have been extensively utilized:

U.S. EPA. 1980a. Ambient Water Quality Criteria Document for Acenaphthene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Water Regulations and Standards, Washington, DC. EPA 440/5-80-015. NTIS PB 81-117269.

U.S. EPA. 1980b. Hazard Profile for Acenaphthene. 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. 1983. Reportable Quantity Document for Acenaphthene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Emergency and Remedial Response, Washington, DC.

ABSTRACT

Because of the lack of data for the carcinogenicity and threshold toxicity of acenaphthene risk assessment values cannot be derived. The ambient water quality criterion of 0.2 mg/ℓ is based on organoleptic data, which has no known relationship to potential human health effects. Acenaphthene has been shown to produce nuclear and cytological changes in microbial and plant species (U.S. EPA, 1980a). Results of acenaphthene mutagenicity studies in microorganisms (Guerin et al., 1978; Douglas et al., 1980) and carcinogenicity study (Neukom, 1974) are negative. Despite the negative results in the newt (Triturus cristatus) the fact that acenaphthene is a PAH, a class of chemicals that contain carcinogens, the carcinogenic potential of acenaphthene is of great concern. Inadequate evidence to allow any conclusion regarding carcinogenicity for humans appropriately places acenaphthene in Group D (U.S. EPA, 1986b).

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LIST OF ABBREVIATIONS

CAS	Chemical Abstract Service
DNA	Deoxyribonucleic acid
PAH	Polynuclear aromatic hydrocarbon

1. ENVIRONMENTAL CHEMISTRY AND FATE

Selected physical and chemical properties and environmental fate of acenaphthene are presented in Table 1-1.

The half-life of acenaphthene in air, water and soil could not be located in the available literature. In both air and water, acenaphthene may be partly associated with particulate matter (Callahan et al., 1979). When adsorbed to particulate matter in air, acenaphthene could potentially be transported long distances before ultimately being removed by chemical reaction or by rainfall and dry deposition (HSDB, 1986; Ligocki et al., 1985). The part of acenaphthene present in the atmosphere in the vapor phase is expected to undergo direct photolysis or oxidation by reaction with photochemically generated hydroxyl radicals (estimated oxidation half-life ~19 hours) (HSDB, 1986; U.S. EPA, 1986a). In water, acenaphthene is expected to be transported primarily as adsorbed matter on suspended solids and may persist for years adsorbed to sediments (Bjoerseth et al., 1979). Ultimate removal by biodegradation may be possible (Callahan et al., 1979). Acenaphthene is expected to strongly adsorb to soil. Ultimate removal by biodegradation is probably more rapid in soil than in water (HSDB, 1986).

According to U.S. EPA (1978), acenaphthene has been detected in effluents from petro-chemical, pesticide and wood preservative industries. Acenaphthene has also been identified in gas exhaust (Grimmer et al., 1977) and cigarette smoke condensates (Harke et al., 1976; Severson et al., 1976). Quantitative data regarding oral and inhalation exposure were not located, but it appears that both routes of exposure may be relevant.

TABLE 1-1

Selected Physical and Chemical Properties and Half-Lives for Acenaphthene

Property	Value	Reference
CAS number:	83-32-9	
Chemical class:	polynuclear aromatic hydrocarbon	
Molecular weight:	154.21	
Vapor pressure at 25°C:	4.47×10^{-3} mm Hg	Mackay and Shui, 1981
Water solubility at 25°C:	3.88 mg/ℓ	Mackay and Shui, 1981
Log octanol/water partition coefficient:	3.92	Hansch and Leo, 1985
Bioconcentration factor:	387 in bluegill sunfish	Barrows et al., 1980
Log soil sorption coefficient:	3.31 estimated	Lyman et al., 1982
	4.27 estimated	Sabljic, 1984
Half-lives in		
Air:	hours to days (vapor phase)	U.S. EPA, 1986a
Water:	years (adsorbed to sediments)	Bjoerseth et al., 1979

2. ABSORPTION FACTORS IN HUMANS AND EXPERIMENTAL ANIMALS

Pertinent data regarding the absorption of acenaphthene following oral or inhalation exposure could not be located in the available literature.

3. TOXICITY IN HUMANS AND EXPERIMENTAL ANIMALS

3.1. SUBCHRONIC

3.1.1. Oral. Knobloch et al. (1969) orally administered 2 g/kg acenaphthene in olive oil to seven young rats (sex not specified) daily for 32 days. The effects observed were loss of body weight, changes in peripheral blood, increased aminotransferase levels in blood serum, mild morphological damage to the liver and kidneys, and mild bronchitis and localized inflammation of the peribronchial tissue. No information was provided regarding the use of controls.

3.1.2. Inhalation. Reshetyuk et al. (1970) exposed 100 rats to acenaphthene at a concentration of 12 ± 1.5 mg/m^3 for 4 hours/day, 6 days/week for 5 months. The authors reported altered reflexes in the upper airways and an increase in the concentration of nucleic acids in the liver. Histopathological examination of the lungs revealed aspecific pneumonia with the bronchial epithelium showing hyperplasia and metaplasia. No signs of malignancy were observed. Further details of this study were not provided.

3.2. CHRONIC

Pertinent data regarding the effects of chronic exposure to acenaphthene following oral or inhalation routes of exposure could not be located in the available literature.

3.3. TERATOGENIC AND OTHER REPRODUCTIVE EFFECTS

Pertinent data regarding teratogenic or reproductive effects of acenaphthene following oral or inhalation exposure could not be located in the available literature.

3.4. TOXICANT INTERACTIONS

Acenaphthene has been shown to depress the activity of dimethylnitrosamine demethylase in rats (Argus et al., 1971; Arcos et al., 1976). This activity is required for carcinogenesis caused by dimethylnitrosamine; therefore, acenaphthene may slightly inhibit dimethylnitrosamine carcinogenesis.

Buu-Hoi and Hien-Do-Phouc (1969) injected male Wistar rats intraperitoneally with 20 mg/kg acenaphthene in corn oil. The rats were injected intraperitoneally 1 week later with 90 mg/kg zoxazolamine. The mean paralysis time of acenaphthene-treated rats was found to be significantly greater ($p<0.01$) than that of vehicle injected rats. The authors believed that these results indicated that acenaphthene slows the detoxification of zoxazolamine, which usually proceeds by hydroxylation.

4. CARCINOGENICITY

4.1. OTHER RELEVANT DATA

Neukomm (1974) reported negative results in a predictive carcinogenicity test based on neoplastic induction in the newt, Triturus cristatus. Acenaphthene was injected subcutaneously at unspecified dose levels.

Mutagenicity studies of acenaphthene in Salmonella typhimurium gave negative results in strain TA98 with and without S-9 metabolic activation (Guerin et al., 1978; Douglas et al., 1980) and in strains TA1535, TA100, TA1537 and TA1538 with S-9 metabolic activation (Douglas et al., 1980). Acenaphthene also had no effect on the recombination rate of two auxotrophic strains of Escherichia coli, as indicated by the low level of prototroph induction (Clark, 1953a). Clark (1953b) also tested acenaphthene for mutagenicity in Micrococcus pyrogens var. aureus strain FDA209 with negative results.

Acenaphthene has been shown to produce nuclear and cytological changes in microbial and plant species. The changes observed, including increased cell size and DNA content, are associated with disruption of the spindle mechanism during mitosis. Because there is no known correlation between these effects and the biological impact of acenaphthene on mammalian cells, studies examining these changes will not be summarized here. Studies concerning these mitotic effects are reviewed in U.S. EPA (1980a).

4.2. WEIGHT OF EVIDENCE

Because of the lack of studies concerning the carcinogenic potential of acenaphthene, the compound can be classified as an IARC Group 3 chemical (inadequate evidence to allow any conclusion regarding carcinogenicity for humans). According to the EPA classification scheme (U.S. EPA, 1986b), acenaphthene is most appropriately included in Group D (not classified).

5. REGULATORY STANDARDS AND CRITERIA

The ambient water quality criterion for acenaphthene, based on organoleptic data, is 0.02 mg/ℓ (U.S. EPA, 1980a). This level has no known relationship to potential human health effects.

6. RECOMMENDATIONS

Because of the lack of data for the carcinogenicity and threshold toxicity of acenaphthene, risk assessment values cannot be derived. The ambient water quality criterion of 0.2 mg/ℓ is based on organoleptic data, which has no known relationship to potential human health effects.

The best documented effect of acenaphthene is its ability to cause nuclear and cytologic changes in plants (U.S. EPA, 1980a). No correlation between these effects and effects on mammalian cells is known. Acenaphthene has tested negative in mutagenicity studies in microorganisms (Guerin et al., 1978; Douglas et al., 1980; Clark, 1953a) and in a carcinogenicity study in the newt, Triturus cristatus (Neukom, 1974). Despite the negative results, the fact that acenaphthene is a PAH, a class of chemicals that contain carcinogens, indicates that the primary issue requiring resolution is the carcinogenicity of acenaphthene by oral or inhalation exposure. Acenaphthene has been found in both air and water (U.S. EPA, 1980a), so that both routes of exposure may be important.

If adequate testing determines that acenaphthene is not carcinogenic, efforts should be made to define thresholds for noncarcinogenic toxicity. Data are needed to determine the target organ(s) or system(s) most likely to be injured by exposure to acenaphthene. Because acenaphthene has a relatively low vapor pressure (4.47×10^{-3} mm Hg at 25°C), substantial levels in air are unlikely and initial testing by oral exposure to determine subchronic, developmental and reproductive toxicity may be more immediately necessary.

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